The Journal

OF THE KANSAS MEDICAL SOCIETY

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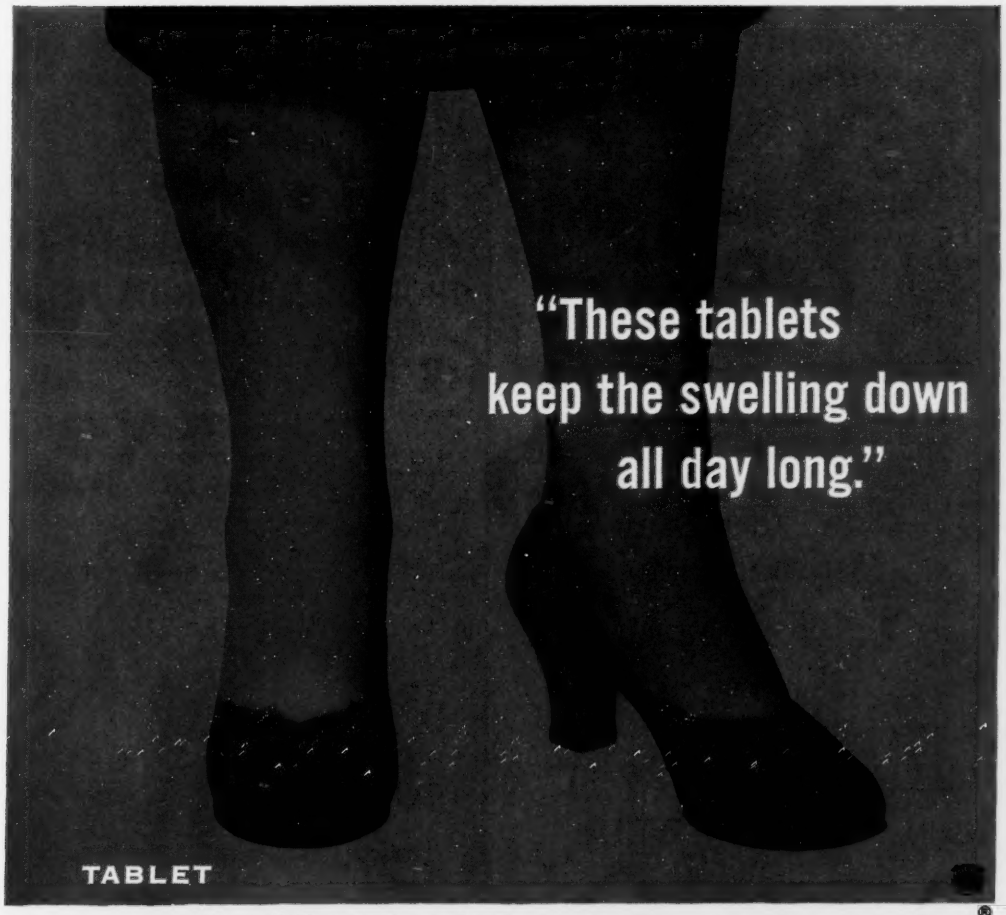

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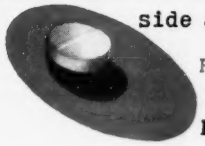

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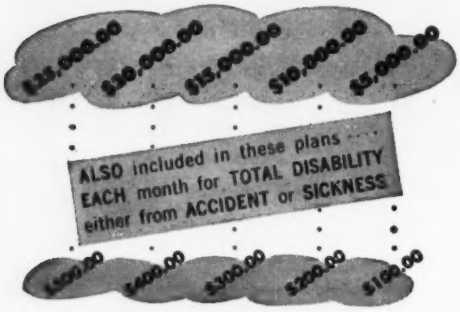

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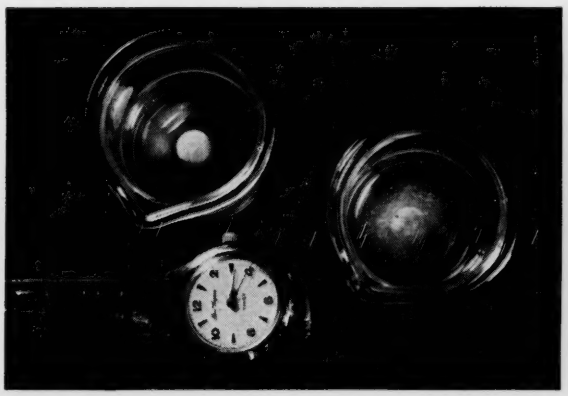

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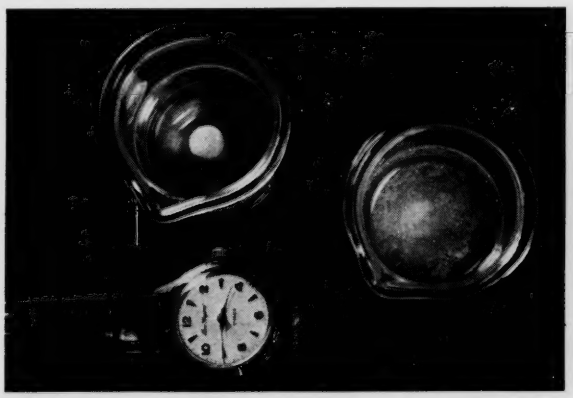

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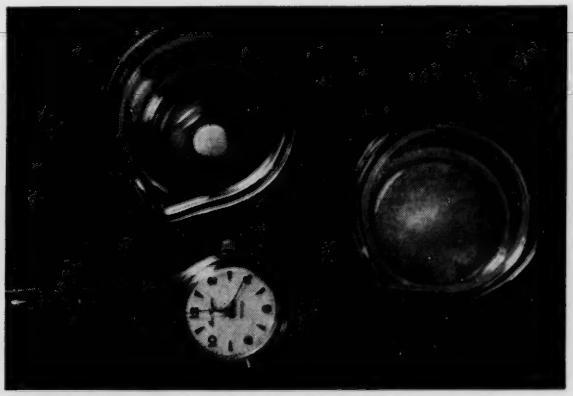

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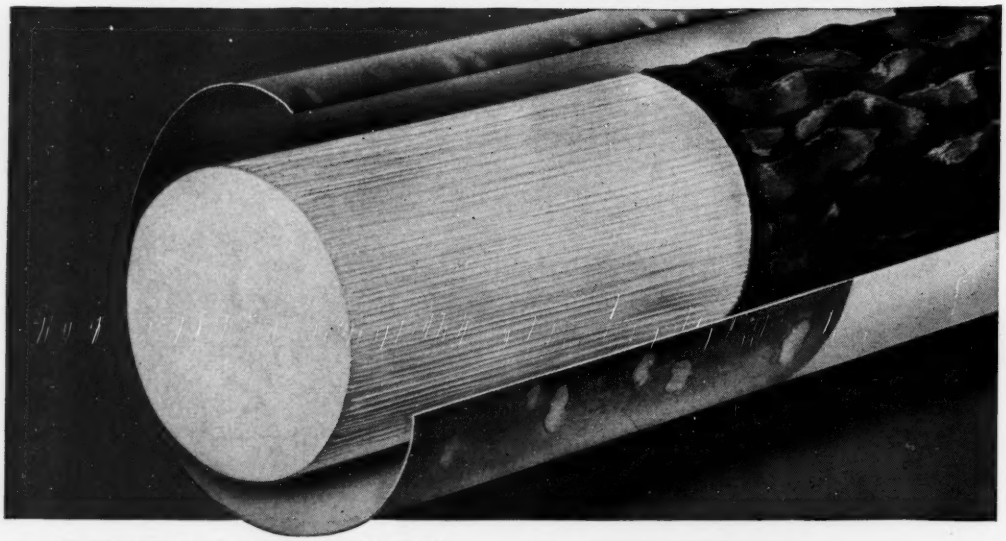

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*Kirsner, J. B.; Brandt, M. B., and Sheffner, A. L.: Diet and Amino Acid Utilization in Gastrointestinal Disorders, J. Am. Dietet. A. 29:1103 (Nov.) 1953.

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1. Kauntze, R., and Trounce, J.: Treatment of Arterial Hypertension with Veriloid (Veratrum Viride), Lancet 2:1002 (Dec. 1) 1951.
2. Wilkins, R. W.: Combination of Drugs in the Treatment of Essential Hypertension, Mississippi Doctor *30*:359 (Apr.) 1953.
3. Stearns, N. S. and Ellis, L. B.: Acute Effects of Intravenous Administration of a Preparation of Veratrum Viride in Patients with Severe Forms of Hypertensive Disease, New England J. Med. *246*:397 (Mar. 13) 1952.
4. Moyer, J. H., and Johnson, I.: Intramuscular Veriloid (Aqueous Solution) As a Hypotensive Agent, Am. J. M. Sc. *226*:477 (Nov.) 1953.

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Volume LV JULY, 1954 No. 7

Carcinoma of the Cervix

L. A. Calkins, M.D.
Kansas City, Kansas

That the cure rate of carcinoma of the cervix has been materially improved in the last 20 years is well shown by Dr. Heyman[1] in his last report. In fact, more than twice as many women are being cured of this disease today as were cured 20 years ago. This improvement has come about in two ways. First, there have been larger amounts of radium made available in better and more applicable forms in treatment centers. Whether the new radioactive isotopes will further improve this situation remains to be seen. Voltage of x-ray therapy machines has been increased, and it is not yet apparent what the optimum voltage will prove to be. Quite certainly, the 250,000 volt machine has produced better results than the 150,000 volt machine formerly used.

The more recent studies of Dr. Franklin L. Payne,[2] Dr. Willis E. Brown,[3] and others give promise of further improvements in methods of radiation therapy. Recent experiments at surgical attack have been enlightening, but, so far, have failed to measure up to the results obtained with radiation therapy. In the small per cent (less than 10 per cent) of radiation failures, a proper field of surgical therapy investigation is being exploited in several places. So far, the results of this therapy, as far as ultimate cure is concerned, have been extremely disappointing. Moreover, the complications and injuries to the urinary and intestinal tracts have been no less than appalling. Whether the present extensive and expensive effort of Dr. Meigs to determine this radiation failure early and, therefore, to afford a better chance of surgical success, will prove fruitful, remains to be seen. Such a method, because of its expense, is prohibitive to most clinics.

Second, the American Cancer Society, by its educational campaign, has also contributed much in the way of getting earlier diagnosis and earlier therapy for such patients. As will be shown later, the cure rate of early carcinoma of the cervix is now 85 per cent to 90 per cent, approximately the cure rate of most benign lesions subjected to surgery. That we will ever achieve such an early diagnosis in all patients now seems utterly impossible. That we can completely avoid the too late and, therefore, far advanced tumor in some patients seems likewise impossible. That the far advanced cases should be seldom seen seems entirely possible.

Another feature of the avoidance of bad results has not been sufficiently emphasized. I refer to the so-called "lag period," which is the length of time elapsing between the first visit of the patient to her doctor and the application of definitive therapy. Twenty years ago, this lag period averaged 13 months. Part of this was due to the fear on the part of the patient that any therapy applied would serve to "spread" the disease rather than cure it. Another part was due to the more or less widespread feeling on the part of the general practitioner, and some specialists, that the situation was a more or less hopeless one, and his consequent failure to urge immediate treatment. Fortunately, this long period of 13 months delay has been reduced to about six months. That it should be further reduced is obvious.

Table I shows experience with those patients coming to the University of Kansas Hospital and treated

From the University of Kansas Medical Center.

TABLE I
CARCINOMA OF THE CERVIX
Incidence

	Stage I	*Stage II*	*Stage III*	*Stage IV*	*Totals*
1929-42	32 (7%)	113 (26%)	190 (44%)	100 (23%)	435
1942-48	44 (18%)	76 (31%)	88 (36%)	36 (15%)	244
1949-53	28 (15%)	62 (34%)	54 (30%)	38 (21%)	182
					861

by the writer or some other member of the full time obstetrics-gynecology staff. The patients were available for follow-up examinations.

We have employed the new International Classification in drawing up these figures, wherein Stage I refers to a tumor confined to the cervix so far as can be told by pelvic examination. Stage II shows some degree of spread of the tumor, but not involving more than the upper third of the vagina and not extending entirely to the pelvic wall. Stage III refers to a tumor involving the parametrium entirely to the pelvic wall or the lower third of the vagina, but not involving the rectum or bladder. If such an involvement occurs, it becomes Stage IV.

It is readily apparent from this table that only one-third of our patients were in the early and, therefore, curable stages previous to 1942; one-half of our patients have been in these more favorable groups since that time. It does seem unfortunate, however, that about one out of every seven patients is still in Stage IV—the hopeless group. That this figure can and should be further reduced is obvious.

We are justifiably proud of our results at the University of Kansas Medical Center when these results are compared with those of other treatment centers in this country (Table II). There will always be some difference of opinion as to the exact stage in which

TABLE II
CARCINOMA OF CERVIX
Results of Treatment

1942-46	*Stage I*	*Stage II*	*Stage III*	*Stage IV*	*All Stages*
Buffalo	159 (70%)	448 (45%)	213 (31%)	154 (16%)	42%
New Orleans	91 (68%)	185 (47%)	389 (25%)	71 (4%)	34%
Sloane & Womens	117 (59%)	137 (30%)	59 (9%)	12 (0)	35%
Jefferson & Penn.	69 (73%)	117 (47%)	87 (23%)	35 (6%)	41%
St. Louis	53 (74%)	92 (42%)	41 (24%)	7 (0)	46%
Stanford & Calif.	121 (67%)	166 (33%)	68 (28%)	18 (0)	42%
Kansas (1942-48)	610 (68%) (84%)	1145 (42%) (58%)	857 (25%) (33%)	297 (9%) (0)	45%

any given patient should be classified when examined by different people. These differences are, however, not nearly so great as formerly. Because of this personal equation, it is argued that over-all rates of cure should be the only consideration, rather than the results in the individual stages of the disease. This is only partially true, since the cure rate in Stage IV is virtually zero, and if one is inflicted with a large proportion of Stage IV patients, he cannot hope to obtain the best over-all rate. The avoidance of late diagnosis and also long lag periods is, therefore, of great importance.

Table III shows the results of therapy over the past

TABLE III
CARCINOMA OF CERVIX
Results of Therapy

	Stage I	*Stage II*	*Stage III*	*Stage IV*	*Totals*
1929-42	32—21 (66%)	113—52 (46%)	190—50 (26%)	100—1 (1%)	435—124 (28%)
1942-48	44—37 (84%)	76—44 (58%)	88—29 (33%)	36—0 (0%)	244—110 (45%)
1949-53	28	62	54	38	182

Too recent to report results.

25 years (except those patients treated in 1949 and later). It is obvious that we have improved our technic and, therefore, our results. A great part of the credit for this improvement should go to Dr. Galen Tice, whose unrelenting efforts at improvement of roentgen therapy have been so important. We are also equally convinced that the accurate placement of radium, with careful individualization of each patient, has paid big dividends. As previously pointed out,[4] careful follow-up with prompt repetitive therapy, when needed, has materially improved our cure rate (4 per cent over-all increase in cures and 10 per cent increase among those requiring additional therapy). This careful follow-up program (only three patients were not followed in the first 500 treated) was started (1) as a labor of love, and (2) to train ourselves in the best possible manner. It has, however, resulted in an increase in cure rate, and it continuously impresses us as being one of the most important items in the treatment of this disease.

The recent report of Dr. Joe Meigs[5] on his experimental surgical approach to early carcinoma of the cervix is worthy of careful consideration. Here we have one of the most expert of all surgeons, selecting only those patients who are good surgical risks, producing results no better than, and sometimes not quite so good as those reported by McKelvey, Norman Miller, and others from radiation therapy, and the series treated by radiation includes patients in all states of physical condition.

It would seem, therefore, (and with this I am sure Dr. Meigs would not disagree) that up to now surgery has less to offer than radiation in the treatment of early carcinoma of the cervix. No one would offer surgery in place of radiation for the later stages. It would seem that the place of surgery, if any, would be in those patients in whom radiation treatment has failed.

The results of the work of Brunschwig, Parsons, and others (pelvic evisceration) are, as yet, not convincing so far as ultimate cure is concerned. The claim that these patients can be temporarily relieved of pain until they die of their disease can well be important. The fact that no expert can always distinguish between the pelvis full of inflammatory induration (so often true of cervical carcinoma both before and after radiation therapy) and the pelvis full of carcinoma, makes this whole problem difficult of adjudication. Many of the evisceration procedures result in removal of inflammatory tissue only—a procedure not so dangerous as it would have been before the antibiotic era. It is obvious that failure of irradiation should not be followed by additional irradiation. It is difficult for the physician to tell such patients that there is no hope and nothing more to be done. Perhaps surgery should be offered to these individuals when death is not imminent. It is equally obvious that such a major surgical procedure as evisceration should not be done on a virtually moribund patient.

The "present status" of treatment of cervical carcinoma would seem to be (1) further education of the public (and physicians) in early diagnosis; (2) further reduction in the lag period between first examination and definitive therapy; (3) application of the most modern methods of pelvic examination, staging of the tumor, accurate radium placement and dosage, and careful, accurately watched, roentgen therapy; (4) meticulous follow-up with possible surgical attack on radiation failures with re-radiation in incomplete results, determined at the end of three to six months and in cases of recurrences discovered as much as 15 years later. We feel that these examinations should be at intervals of several weeks for the first two years, less frequently for the next three years, with one or two examinations per year thereafter. Urinary tract evaluations and chest x-rays should be made on slight indications. We are strongly of the opinion that all these patients should be followed for a minimum period of 15 years.

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Spontaneous Intravascular Clotting

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Discussions on increased coagulability of intravascular blood are few. In the cases reported, the condition exists usually secondary to some other condition, such as phlebothrombosis following sudden or prolonged rest incident to myocardial infarction, or by pelvic (or lower portion of the body) surgery, or by trauma. Inflammatory conditions as thrombophlebitis and septic emboli have been held to be etiological agents in intravascular clotting.

Recently there have been noted cases of increased coagulability of the blood of patients who have acute pancreatitis. The phenomena which result are different from those in patients in whom there are infarctions, or those in whom circulatory stasis of the extremities tends toward the production of thromboembolism. There are produced changes in the blood itself, brought on by the injection into or absorption of humoral agents from a diseased organ of the body.

Pathologically, in acute pancreatitis there is an edematous pancreas, swollen, and with much hemorrhage and hyperemia throughout. There are usually numerous areas of necrosis of the fat in and immediately adjacent to the gland, and there may be an associated chemical peritonitis, with or without bloody discoloration. Microscopically, there appears to be a metaplasia of the epithelial cells of the pancreatic ducts, the etiology of which seems to be obscure and unproved, plus areas of polymorphonuclear infiltration of the gland. Factors such as chronic alcoholism,[1] regurgitation of infected bile into the pancreatic ducts following obstruction of the latter at the ampulla of Vater (or regurgitation of food into the accessory pancreatic duct), or even a humoral agent, as from acute parotitis, have been implicated in the cause, and cases have been published which have actually shown the apparent connection of the alleged inciting agent and the pancreatitis. It is prob-

able that there is no one of these that has more merit than another, or it is possible that the actual cause has not been found at all.

In its pathologic physiology, the destruction of the gland allows some of the secretions, such as trypsin, etc., to directly enter the blood stream, where it affects the various components already present and elicits an anaphylactic response producing shock.[2] Prothrombin is converted to thrombin, resulting in increased coagulability of the blood intravascularly. It is thought also that trypsin inhibits the action of heparin, thus allowing the clotting of blood to occur more freely. (Paradoxically, there has appeared in the literature recently the treatment of thrombophlebitis with intravenous instillations of trypsin.)[3]

In the cases presented, there are shown the findings of massive intravascular clotting, both venous and arterial, with its profound attendant phenomena and sequellae.

Case No. 1, Providence Hospital No. 53-1004, Mrs. N. A., age 62. The illness of the patient began on January 30, 1953, with the complaint of shortness of breath on exertion and nausea of one day's duration. The patient had arteriosclerotic heart disease and arterial hypertension, and her blood pressure that date was 190/102 mm. Hg. Other physical findings that date were auricular fibrillation and extreme obesity. The chest was clear, abdomen was obese, and there was tenderness on palpation in the right upper quadrant. She was seen again at the office on February 3, 1953, because of severe nausea unproductive of vomitus. She apparently improved, and nothing was heard of her until February 11, 1953, when a call was received from a member of the family who stated that the patient had become mentally confused.

On hospitalization that date, she was unable to give a satisfactory history. She complained then of a tenderness behind her right knee. There was a temperature of 101°F.; she was irrational and uncooperative. Blood pressure was 170/100 mm. Hg., pulse was 96, respirations 20, heart rhythm grossly irregular, abdominal obesity, with slight tenderness in the right upper quadrant. There appeared to be a swelling in the popliteal area of the right lower extremity.

Laboratory findings on February 12, 1953, showed the urine to have a specific gravity of 1.020, with a trace of albumin, negative sugar, and 8 to 10 fine and coarse granular casts. The hemoglobin was 13.5 gms., or 87 per cent; the red blood count was 4.48 million. The white blood count was 13,700 with 81 per cent polymorphonuclears, 78 of which were filamented and 3 non-filamented, 16 per cent lymphocytes, 2 per cent monocytes, and 1 per cent basophiles. The sedimentation rate was 16 mm. in 1 hour. On February 21, 1953, the serology was 3 plus. On February 23, 1953, the white blood count was 20,000 with 90 per cent polymorphonuclears. On February 26, 1953, the prothrombin time was control 12.5", patient 26", 19 per cent prothrombin activity.

Roentgenological examination of the chest (portable) on February 12, 1953, showed an elevation of the right diaphragm (slight), no evidence of pulmonary parenchymal infiltration, cardiac size and contour average normal, with the roentgenologist's comment that there was no gross evidence of disease. Electrocardiographic studies this same date showed auricular fibrillation; a repeat electrocardiogram on February 20, 1953, again showed auricular fibrillation, plus bradycardia due to digitalis and the possibility of some disturbance in the anterior coronary artery.

Progress in the Hospital: The mental confusion gradually cleared. She ran a spike-type temperature with daily elevations of up to 101°F., and 102°F. The day after admission the veins behind the right knee became swollen, and cold compresses were applied to the area. She was given 500,000 units of penicillin intramuscularly twice daily, in addition to daily depo-heparin. On February 16, 1953, digitalis was discontinued and quinidine therapy was started in an attempt to control the auricular fibrillation which was thought to be sending off showers of emboli into the circulation. There was noted a beginning cyanosis of the right foot up to the ankle on February 21, 1953. Her blood pressure that date was 126/70 mm. Hg. The next day there was noted extension of the cyanosis to the right forearm and left leg.

On February 24, 1953, the temperature was normal. The patient stated that she felt better, in spite of the condition of her arm and legs. Surgical consultation result was "saddle embolus at bifurcation of the aorta"; medical consultation result was that the patient was having "multpile emboli and arterial occlusion, with possible secondary thrombi, with her cardiac condition preventing surgery (embolectomy)."

The patient expired on February 27, 1953. The pertinent findings at autopsy were: The body was that of an approximately 60-year-old, obese white female 5 feet 10 inches tall weighing approximately 200 pounds. Head, neck and chest showed no abnormalities. The abdomen was moderately distended; no masses could be felt. The lower extremities showed gangrene with swelling and redness extending to an area 6 cm. above the knee.

In section, the heart showed a subendocardial hemorrhage 3 mm. in diameter on the posterior surface of the left ventricle. Atherosclerosis of the coronary arteries, most marked in the descending branch of the left coronary, was present. The lungs presented a thrombus in a blood vessel of the posterior right lower lobe, and the left lower lobe had

numerous vessels filled with antemortem thrombi that completely occluded the vessels. Microscopically, no attachment of the thrombus could be demonstrated. The liver, gall bladder, spleen, kidneys, head, neck, and thyroid showed nothing of note. The pancreas showed areas of yellowish fatty degeneration with focal areas of necrosis. Zones of hemorrhage surrounded some of the necrotic areas. In some instances a purulent appearing material could be expressed from the cut surface. The necrotic lesions were focal but were diffusely spread throughout the pancreatic tissue. No obstruction to the pancreatic ducts could be identified. Microscopically the acinar cells were moderately retracted from their surrounding basement membrane. Most sections contained large areas of fat necrosis with a cellular debris at the periphery in some and an infiltration of lymphocytic cells, along with an occasional polymorphonuclear leukocyte, in others. There was an increase of fibrous tissue surrounding some of the areas of necrosis with proliferation of fibroblasts. In still other sections, the areas of fat necrosis were surrounded by an inflammatory reaction with no fibroblastic proliferation. In several of the sections, hemorrhage was seen in the tissue surrounding the areas of fat necrosis.

The aorta was opened throughout. The wall was of normal thickness. Several small linear lipoidal streaks were identified, but otherwise no unusual changes were seen. The iliac vessels were examined, and a thrombus was present in the left iliac artery beginning at a distance of 5 cm. from the bifurcation of the aorta. The thrombus extended for a distance of 5 cm. into the femoral artery. The lumen in this area was dilated; the walls showed no gross change. The external saphenous, the iliac and the femoral veins, and the popliteal veins contained an antemortem thrombus. The right femoral vein similarly had an antemortem thrombus. At a distance of 1 cm. from the origin of the supraclavicular artery, an antemortem thrombus was present in the brachial artery that completely occluded the vessel.

In one of the sections of the adrenal, a zone of necrosis was noted into which hemorrhage had occurred. In another section the architecture of the cortex was disturbed and replaced by a group of cells which were well circumscribed from the surrounding adrenal parenchyma. These cells had a foamy cytoplasm with basophilic nuclei.

Microscopic sections of the cervix, Fallopian tubes, ovaries, brain, pituitary, and thyroid showed nothing pertinent.

The final diagnosis was: (1) acute fat necrosis of the pancreas; (2) thrombosis of both iliac and femoral veins, the left iliac and femoral arteries, and the right brachial vein artery; (3) gangrene of the distal one-third of both thighs and lower legs; (4) fatal massive pulmonary embolism, left, with areas of infarction of the lower lobe of the left lung; (5) coronary arteriosclerosis; (6) hyperplasia of the right adrenal cortex.

Here was a case of acute pancreatitis that had nausea as the presenting symptom. There was never abdominal pain, and the patient complained of tenderness in the right upper quadrant on deep palpation only. She later complained of swelling behind the right knee, was seen to have gangrene of the

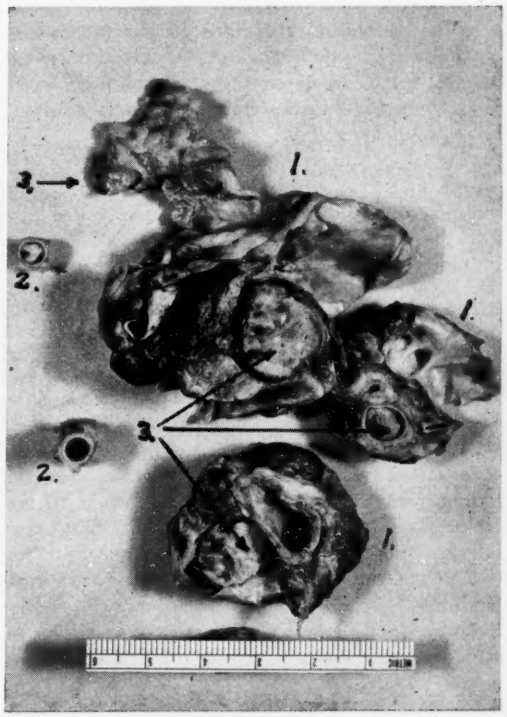

Figure 1. Case 1. Tissue. 1—lung; 2—peripheral arteries with thrombi; 3—thrombi.

extremities, and autopsy showed antemortem thrombi in both arterial and venous vessels. The fact that the coagulation occurred in both arteries and veins shows that a change in the consistency of the blood had occurred so that spontaneous clotting within the vessels took place in different parts of the body simultaneously.

Case No. 2, Providence Hospital No. 54-1382, H. H., age 55. The patient was a male in good nutritional state. On February 17, 1954, the patient called on the phone and stated that he had severe pain in his right upper side; he requested that arrangements be made to have his gall bladder "cut out." He was advised that x-ray pictures and examinations be made to be sure of the necessity of such a procedure, and on February 18, 1954, roentgenological examination

showed a normally functioning gall bladder without stones.

Nothing more was heard of the patient until February 23, 1954, when he again phoned and complained of a constant pain in his right upper abdomen. He stated that he had vomited "a bitter material" a few times. I advised hospitalization, and further history at the hospital revealed that he had had stomach trouble for some years, and that he dated his present condition back 10 days when he noticed his "abdomen beginning to swell."

In the past history there was no gallbladder disease, no ulcers, no alcoholism. Further history, by systems, revaled some shortness of breath on exertion. There was no history of diarrhea, constipation, or genitourinary disease.

Physical examination on admission revealed the patient to be dehydrated, sitting up in bed, with shortness of breath and with severe abdominal distention. He stated that he felt better when he was in sitting-up position. The abdomen revealed a board-like rigidity in the right gastric area and in the right hypochondrium.

The admitting laboratory reports were: urine: color straw, reaction pH 6.0, specific gravity -1023, albumin faint trace, sugar 0, microscopic negative. The red blood count was 5,080,000, with 14.6 gm.—94.2 per cent of hemoglobin. The white blood count was 10,000, with 89 per cent polymorphonuclear cells, filamented 79 per cent, non-filamented 10 per cent, and 11 per cent lymphocytes. Non-protein nitrogen was 32 mg. per cent. The serum bilirubin was .? mg. per cent "direct" and .3 mg. per cent "indirect." Blood amylase was 42 units. The prothrombin time showed 60 per cent of prothrombin activity. Lipase was 0.3 cc. The sugar tolerance test was:

Fasting 158 mg. Urine neg. 2 hours 235 mg. Urine 4 plus
½ hour 277 mg. Urine neg. 3 hours 175 mg. Urine 4 plus
1 hour 293 mg. Urine 4 plus 4 hours 153 mg. Urine 1 plus

Progress: The patient complained considerably of steady pain in his abdomen; this pain was non-radiating. A Wangensteen tube was inserted because of distention and vomiting. He was given an intravenous solution of 2,000 cc. of 5 per cent glucose in saline and pencillin, one million units plus one gram of streptomycin daily. Demerol, 75 mg., with 1/150 grain of atropine was given for pain. In addition, he was given transfusions.

With the negative laboratory findings to back up a diagnosis of acute pancreatitis (with the exception of the blood sugars), he was taken to surgery for surgical decompression (Dr. M. A. Walker). With the advice of the hospital roentgenologist, who stated that there was a possible obstruction at the splenic flexure, a cecostomy was done for decompression. This was without results, and it was felt then that there may have been a small bowel obstruction instead. The abdomen postoperatively remained distended. The cecostomy was irrigated every six hours. The penicillin-streptomycin injections were continued. The fluid from the Wangensteen came back brownish red, as also did the fluid from the cecostomy. On March 3, 1954, the non-protein nitrogen was 41 mg. per cent. The total proteins on this date were 5.90; albumin 4.05, globulin 1.85.

On March 8, 1954, a repeat roentgenogram of the abdomen showed the small intestines to be filled with gas, suggesting the presence of generalized peritonitis. The roentgenologist stated that the impression was that of ileus, and there appeared to be some fluid in the abdominal cavity. Clinically the patient's pulse ran between 90 and 130, being elevated most of the time, and his temperature was between 102° and 104°.

The condition of the patient deteriorated rapidly; however, he remained conscious and rational. On March 8, 1954, the non-protein nitrogen was 65. Total proteins were 7.40; albumin 3.30, globulin 4.10. The blood sodium which was 144.2 mEq/1 on March 3, 1954, had elevated to 151 mEq/1. Potassium showed no changes as evidenced by the electrocardiogram which also showed a normal sinus rhythm. However, he was given potassium chloride, 20 cc. (2.98 gm.), one ampule, through the nasal tube.

The patient expired on March 9, 1954. The autopsy, with the body being opened with the usual Y-shaped incision, showed the folowing: The peritoneal surfaces were smooth with the exception of the omentum and loops of intestine adherent by fibrinous adhesions to the surgical incision in the right lower quadrant. Adhesions were also present between several loops of bowel and the gallbladder. Heart showed mild atherosclerosis with mild narrowing of the coronary lumina. No thrombi were present. The lungs showed red thrombi in the smaller vascular channels of the left lower lobe. The right lower lobe was firmer in consistency, dark red in color, and on section showed extensive congestion and interstitial hemorrhage. The larger vessels were dilated and contained red thrombi. Histologically, a section through a large pulmonary artery, dilated and containing a red thrombus, was taken. The adjacent pulmonary parenchyma showed extensive edema and hemorrhage in the alveolar spaces with congestion of the alveolar walls. The liver showed nothing of note. The gallbladder showed an inflammatory process, with a lymphocytic infiltration in the mucosa and through the wall. There was also seen necrosis of the overlying epithelial folds with deposition of brownish bile pigment within the necrotic debris. Large foamy phagocytes were present. The extreme

edge of the section showed necrosis of the entire wall with polymorphonuclear leukocytes and a heavy lymphocytic infiltration. The spleen showed nothing abnormal. The pancreas showed areas of moderate increase in fibrous tissue and focal areas of lymphocytic infiltration. In many sections, focal areas of necrosis were present involving pancreatic tissue and the surrounding fat. These areas showed a bluish discoloration with loss of the architecture. In some, an infiltration of polymorphonuclear leukocytes was seen in the adjacent fat tissue with lymphocytes more peripherally. The adrenal, kidneys, bladder, prostate, and gastrointestinal tract showed nothing unusual. The aorta showed thickening of the intima.

The final diagnosis was: (1) acute pancreatic fat necrosis with acute peritonitis, adynamic ileus (first portion of small intestine) and necrosis of the wall of the gall bladder; (2) pulmonary thrombosis with infarction of the lung; (3) recent cecostomy; (4) generalized arteriosclerosis with calcific aortic arteriosclerosis and mild coronary arteriosclerosis; (5) generalized visceral congestion.

The comment by the pathologist was that this patient had a thrombosis in the pulmonary vessels and/or emboli of the same material and subsequent infarction.

Diagnosis of acute pancreatitis is becoming relatively more common. The usual picture of the patient in extreme upper abdominal pain (the patient of Case No. 1 had no pain, but had extreme nausea; it is possible that she felt little pain because of shock), the presence of shock, cyanosis, low blood pressure, rapid pulse and distended abdomen produced by ileus, as was shown in Case No. 2, point to the possibility of acute pancreatitis. Laboratory aids are important, but one must consider the timing and clinical value of the tests. Raffensperger showed that dependence on elevated serum amylase as a diagnostic criterion could be erroneous as various other factors could produce the elevation, namely, peptic ulcer, acute peritonitis, edema of the outlet of the pancreatic ducts, and small bowel obstruction.[4] The amylase test is of real value only in the first 24 hours. Most of the tests shown in the second case were normal with the exception of the glucose tolerance test, which showed an evident damage to the pancreas.

SUMMARY

The additional finding of thrombosis occurring intravascularly in cases of acute pancreatitis is of considerable value, and this should increase our efficiency in diagnosis. The presence of thrombosis in both the venous and the arterial systems simultaneously, early detection of cyanosis or even beginning gangrene of the extremities point to the existence of a change in the character of the blood itself which could have its origin in the pancreas.

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The true greatness and the true happiness of a country consist in wisdom, in that enlarged and comprehensive wisdom which includes education, knowledge, religion, virtue, freedom, with every influence which advances and every institution which supports them.

—*Henry Giles*

Poisonings with the Newer Pesticide Compounds

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In the past few years a large number of new pesticides have been introduced and widely used in agriculture, industry, and the home. Many of these pesticides are highly toxic. The problem of identification and treatment is often difficult because of the complicated formulations in which specific toxicants may be found. Kansas law requires the registration of formulas of all pesticides sold in the state. These formulas are on file in the Control Division of the State Board of Agriculture in Topeka. In cases of poisoning where a trade name of a pesticide is known, a call to this department will permit the physician to obtain complete information on the toxicants. If the original container can be located, the label should give a complete formula.

In many cases of poisoning, the nature of the toxic agent or agents is not known. The treatment is, therefore, symptomatic and may include:

1. Emesis and gastric lavage if poison has been taken internally.

2. Evacuation of the gut (avoiding oily laxatives where it is possible that an organic solvent or a halogenated insecticide is involved).

3. Thorough washing of the eyes or body if there has been external contact with the poison—wear rubber gloves while aiding the patient.

4. Supportive therapy.

A. Sedatives. Sodium pentobarbital is preferred for acute poisoning because of its rapidity of action. Phenobarbital is useful in maintaining a prolonged level of sedation in persisting hyperexcitability or convulsions.

B. Stimulants. In treating vascular collapse, substances such as adrenalin should be used only after careful consideration, as they are contraindicated in poisoning by the halogenated hydrocarbons.

C. Transfusions. In patients *in extremis* or extended syncope, transfusions may be indicated except where pulmonary edema is already present. If blood is not readily available or if dehydration is suspected, 5 per cent glucose or normal saline infusions are indicated.

D. Oxygen therapy. Conventional oxygen-carbon dioxide mixtures should be administered to patients showing cyanosis or severe respiratory difficulty. Patients with pulmonary edema require oxygen under positive pressure as well as postural drainage and dehydration therapy until the exudate is checked.

Dr. Hood is executive secretary of the Kansas State Board of Health. Mr. Wright, also with the Board, is director of the Food and Drug Division.

INSECTICIDE FORMULATIONS

Insecticides for agricultural and industrial use may be prepared in many ways. They may be the pure toxicant substance in liquid, crystalline, or powder form. They may be solutions in water or hydrocarbon solvents, emulsions, dusts, wettable powders, or suspensions. Household insecticides may be powders, dusts, solutions in either water or hydrocarbons. The popular "bug bombs" are solutions of the toxicants in hydrocarbons, usually deodorized kerosene, with a Freon propellant to produce an aerosol. Synergists or activators are often added to the formulations to give greater killing power.

For greater convenience, the most common of the newer insecticides may be divided into two types—the chlorinated hydrocarbons and the organic phosphates.

THE CHLORINATED HYDROCARBONS

DDT	Dichloro-diphenyl-trichloroethane
TDE	Dichloro-diphenyl-dichloroethane
DFDT	Difluoro-diphenyl-trichloroethane
Methoxychlor	Dimethoxy-diphenyl-trichloroethane
BHC	Technical benzene hexachloride
Lindane	Gamma isomer of benzene hexachloride
Toxaphene	Chlorinated camphene
Chlordane	1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
Aldrin	1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-hexahydro-1,4,5,8-dimethanonaphthalene
Dieldrin	1,2,3,4,10,10-Hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-dimethanonaphthalene
Heptachlor	1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene
Prolan	2-Nitro-1,1-bis(p-chlorophenyl) propane
Bulan	2-Nitro-1,1-bis(p-chlorophenyl) butane

Routes of Absorption: All the chlorinated hydrocarbons are absorbed through the skin as well as from the gastrointestinal system and respiratory system. The compounds which are normally liquids are in general absorbed through the skin more rapidly than those which are normally solids. Solutions in oily or hydrocarbon solvents are more actively absorbed than dry formulations.

Symptoms: In general the chlorinated hydrocarbons are central nervous system stimulants. The mechanism of their action is not known. Symptoms most frequently experienced are hyper-excitability, irritability, loss of equilibrium, clonic-tonic convulsions. Depression sometimes follows other symptoms. Loss of appetite is a common symptom.

Treatment: Treatment is essentially the same for poisoning by any of the chlorinated hydrocarbons.

Wash the contaminated skin thoroughly with soap and water. Avoid cleaning skin areas with such materials as benzene, gasoline, or carbon tetrachloride. Clear the alimentary tract if the poison has been taken internally, using gastric lavage and saline laxatives and avoiding oily laxatives. Sedation to restore a relative calm is necessary, the drugs of choice being sodium phenobarbital, phenobarbital, and pentobarbital. Paraldehyde and urethane have been used effectively. The object of sedation is not to induce sleep but to counteract excitability, and it should not be carried to the extent of interfering with eating. However, large doses of the drugs may be given and pushed to the limit of therapeutic effectiveness. Persons poisoned by stimulants may tolerate large doses of barbiturates without undue depression and with great benefit. Calcium gluconate, intravenously, has been reported to have some effectiveness as an antidote, and, as its mode of action is different, it may be used in conjunction with barbiturate therapy.

THE ORGANIC PHOSPHATES

Parathion	O,O-Diethyl-p-nitrophenyl-thiophosphate
Dimethyl parathion	O,O-Dimethyl-p-nitrophenyl-thiophosphate
Paraoxone	O,O-Diethyl-p-nitrophenyl-phosphate
OMPA	Octamethyl pyrophosphoramide
TEPP	Tetraethylpyrophosphate
HETP	Hexaethyltetraphosphate
ASP-47. Sulfatep	Tetraethyldithionopyrophosphate
Malathion	O,O-Dimethyl dithiophosphate

The uses of the organic phosphates are almost entirely agricultural, although a small amount of parathion has been sold for domestic garden use.

Routes of Absorption: The organic phosphates are readily absorbed through the skin. They may be absorbed through the respiratory system from the inhalation of sprays. Ingestion would be equally dangerous. Most of the poisonings which have occurred have been through spillage of the concentrated chemicals on the skin, or through combined skin exposure and inhalation of sprays. The organic phosphates act as irreversible inhibitors of the enzyme, cholinesterase, and thus allow the accumulation of large amounts of acetylcholine. When the critical level of cholinesterase depletion is reached, symptoms appear. Recovery is apparently complete if there is time for the reformation of the critical quota of cholinesterase; however, additional exposure before recovery is complete has an additive effect.

Symptoms: Symptoms of organic phosphate poisoning include headache, giddiness, blurred vision, weakness, nausea, cramps, diarrhea, and discomfort in the chest. Signs include sweating, miosis, salivation, pulmonary edema, cyanosis, papilledema, convulsions, coma, and loss of reflexes and sphincter control. The last several signs are seen only in advanced cases but do not preclude a favorable outcome if energetic treatment is continued. The onset of symptoms may be quite rapid, and treatment should begin as soon as exposure is recognized.

Treatment: Keep the patient completely atropinized if symptoms appear. Give 1 to 2 mg. (1/60 to 1/30 grain) per hour up to 10 or 20 mg. in a day, particularly for the control of respiratory symptoms. The intravenous route is most rapid. It will be noted that this dosage of atropine is greater than that ordinarily employed for other purposes. Morphine, theophylline, and aminophylline are contraindicated.

If the patient has not yet shown symptoms or if they have been allayed by the first dose of atropine, he should be completely and quickly decontaminated. Wearing rubber gloves, remove the patient's clothing, and with due regard for his condition at the moment, bathe him thoroughly with soap and water. If washing soda is available, use it, for the organic phosphates are hydrolyzed more rapidly in the presence of alkali.

If there is any suspicion that an organic phosphate has been ingested, induce vomiting, give some neutral material such as water, and induce vomiting again.

If pulmonary secretions have accumulated before atropine has become effective, the patient should be turned to a suitable position for postural drainage of mucus. Suction should be used as needed to remove secretions. If the stomach is distended, it may be emptied with a Levine tube.

The use of an oxygen tent or even of oxygen under slight positive pressure is advisable and should be started early. The patient should be watched constantly for the appearance of muscular weakness, and the need of artificial respiration may appear abruptly. Equipment for artificial respiration should be placed at the patient's bed in readiness as soon as he is hospitalized. Complete recovery may occur even after many hours of artificial respiration have been necessary.

The acute emergency lasts 24 to 48 hours, and the patient must be watched constantly during that time. Favorable response to one or more doses of atropine does not guarantee against sudden and fatal relapse. Medication must be continued through the entire emergency.

Following exposure heavy enough to produce symptoms, further organic phosphate insecticide exposure should be avoided. The patient remains susceptible to relatively small exposures of the phosphates until regeneration of the cholinesterase is complete.

THE HYDROCARBON SOLVENTS

Kerosene, deobase, coal oil.—Kerosene is a mixture of aliphatic hydrocarbons distilled from petroleum in the temperature range of 204 to 315 degrees F.

Toluene, toluol, methylbenzene.

Xylene, Xylol, dimethylbenzene—A mixture of *o-*, *m-* and *p-* xylene.

Kerosene, particularly the deodorized type (deobase) is used extensively as the solvent and vehicle for insecticides in household flysprays. It is also used as a solvent in agricultural sprays and bases for emulsions. Xylene is a common solvent in insecticidal solutions, emulsion concentrates, and emulsions. Toluene is used to a lesser extent than xylene for the same general purposes.

Routes of Absorption and Symptoms: In any poisoning resulting from a formulation of insecticide containing any of the hydrocarbon solvents, greater danger to the patient may lie in the solvent rather than in the solute toxicants. This is particularly true if any of the material has been aspirated. In the case of kerosene, it has been shown that absorption from the gastrointestinal tract is not significant as a cause of mortality, but that the small quantity necessary to produce fatal poisoning by aspiration should be emphasized. Ingestion frequently results in immediate gagging and coughing with consequent aspiration of the oil. The initial symptoms are followed by deep drowsiness. In more serious cases, bronchopneumonia may develop in 24 to 36 hours. Xylene and toluene are apparently absorbed from the intestinal tract, and because these substances in technical grades usually contain some benzene, it must be considered that either ingestion or inhalation may affect the hemopoietic system as benzene.

The use of sprays made with hydrocarbon solvents in closed spaces may lead to headache, disturbances of vision, dizziness, poor coordination, and nausea. More severe exposure may result in collapse and coma before the patient is aware of his condition.

All the hydrocarbon solvents are irritating to tender skin and other tissues. Absorption through the skin is not significant under ordinary conditions of exposure. Xylene is particularly irritating to tender skin and to the eyes.

Treatment: If eyes and skin are contaminated, they should be thoroughly washed. Two per cent butyn ointment may be placed in the eyes, and any analgesic ointment may be used on irritated skin areas. If solvent has been ingested, an effort should be made to remove it by gastric lavage. In case of kerosene this is a precaution against regurgitation and aspiration, rather than to prevent absorption from the gastrointestinal tract. Saline laxatives may be used, but oily laxatives should be avoided, especially if the ingested material contained any insecticides. Sedatives or stimulants may be used symptomatically in moderation. Chemotherapy does not benefit kerosene pneumonia but may be useful in preventing bacterial invasion. Liver damage may be minimized by the use of a low-fat, high-protein diet.

Before leaving the field of insecticides, it should be pointed out that many of the old insecticides, particularly compounds of mercury, lead, arsenic, and fluorine, are still used rather widely. Further, there are a number of new insecticides and adjuvants which have toxicity somewhat lower than those mentioned above, but which, under unusual circumstances, cause poisoning. These include the organic thiocyanates.

RODENTICIDES

Several effective rodenticides have been developed in the past few years. They are sold under trade or coined names: 1080, warfarin, pival, castrix, and Antu. These products have almost entirely replaced the phosphorus, arsenic and strychnine, and squill compounds formerly used to poison rodents.

1080, Ten Eighty—Sodium Monofluoracetate. Ten eighty is extremely toxic to all forms of animal life. Because of its great toxicity it is not available for domestic use. It is available in Kansas only to licensed pest control operators placed under bond.

Routes of Absorption and Symptoms: Ten eighty is not readily absorbed through the skin, but dusts of the crystalline material are readily absorbed through the respiratory system. Absorption is complete through the gastrointestinal tract on ingestion of solutions.

Ten eighty acts through direct interference with the acetate metabolism and has a strong effect on the cardiovascular and nervous systems. Symptoms appear in from 30 minutes to 2 hours or more after ingestion. The first indications of poisoning are nausea and mental apprehension followed by epileptiform convulsions. After a period of several hours, pulsus alternans may appear followed by ventricular fibrillation and death. Children appear to be more subject to cardiac arrest than to ventricular fibrillation.

Treatment: Immediate emesis and gastric lavage followed by oral doses of magnesium sulfate are useful. Administration of certain substances capable of supplying acetate ions has shown antidotal effects in animals, including monkeys; the choice drugs are monoacetin (glycerol monoacetate) (2-4 gm./kg.) and a combination of sodium acetate and ethanol (2 gm./kg. of each). A single dose of magnesium sulfate (800 mg./kg.) given intramuscularly as a 50 per cent solution has saved the life of rats dosed with lethal amounts of 1080. Complete quiet and rest are indicated, but barbiturate anesthesia has proved disappointing when used as an antidote against this poison.

Warfarin, Compound 42, WARF-42 — 3-(α-phenyl-β-acetylethyl)-4-hydroxycoumarin

Pival — 2-pivalyl-1, 3-indandione

Routes of Absorption: Warfarin and pival are readily absorbed from the intestine, and apparently no other route is important. Both rodenticides are

essentially anticoagulants. They inhibit the formulation of prothrombin and apparently produce some capillary damage so that the slightest trauma results in hemorrhage. A single dose probably could not be fatal to man, but several small doses over several days would produce death. Poisoning could not be expected from these rodenticides except where suicide or homicide is intended. The action of these rodenticides is similar to that of Dicumarol, except that capillary damage (the so-called "toxic factor") is greatly increased.

Treatment: After blood has been taken for prothrombin and other tests, a blood transfusion should be given at once if there is reasonable assurance that warfarin or pival poisoning has occurred, irrespective of whether or not signs and symptoms are present. Vitamin K in a dose of 65 mgs. (gr.i) repeated three times on the first day of treatment is suggested, irrespective of symptoms, and smaller doses should continue until prothrombin time has reached normal. In a more seriously ill patient, small transfusions of carefully matched whole blood should be given daily until the patient returns to normal.

The progress of the patient should be followed by the prothrombin test. Tests should be made at least twice daily until a return to normal is clearly established.

Antu.—Alpha-naphthylthiourea.

Antu has now been replaced to a large degree by the safer warfarin and pival. It is still a constituent of some rodenticides used for household rodent control.

Routes of Absorption and Symptoms: Antu has no significant toxicity except when ingested. It kills by the production of pleural effusions, by an action not presently known. It has an emetic action, and normally most of the material will be vomited before serious poisoning occurs. It is especially toxic to rats because they cannot vomit.

Treatment: No antidote is known for Antu. Treatment must be entirely symptomatic.

SUMMARY

This article was assembled for publication with the thought that data relating to clinical aspects of toxic pesticides, rodenticides, and solvents might be a useful reference in the hands of practicing physicians.

Acknowledgment: The material for this paper was derived from a number of sources, many of them manufacturers' information and instruction sheets. Some of this information is so general in distribution it cannot be identified as to its original sources. Special acknowledgment should, however, be made of the use of material from Clinical Memoranda on Economic Poisons, Communicable Disease Center, United States Public Health Service, Savannah, Georgia, and the following series of papers published in the Quarterly Bulletin of The Association of Food and Drug Officials of the United States:

Toxicology of the newer agricultural chemicals, by Arnold J. Lehman, M.D., XII (July) 1948.

Pharmacological considerations of insecticides, by Arnold J. Lehman, M.D., XIII (April) 1949.

Chemicals in foods: a report to the Association of Food and Drug Officials on current developments, by Arnold J. Lehman. M.D.: Part II, Pesticides, XV (October) 1951; Part II, Pesticides. Sec. II, Dermal Toxicity, XVI (January) 1952; Part II, Pesticides. Sec. III, Subacute & Chronic Toxicity, XVI (April) 1952; Part II, Pesticides. Sec. IV, Biochemistry, XVI (July) 1952; Part II, Pesticides. Sec. V, Pathology, XVI (October) 1952.

Pesticides: some chemical considerations and toxicological interpretations, by W. I. Patterson and A. J. Lehman, M.D., XVII (January) 1953.

Distribution and adsorption characteristics of vaporized lindane, by W. A. Queen, XVII (October) 1953.

Toxicological evaluation of household insecticides, by Arnold J. Lehman, XVIII (January) 1954.

The educational process is a universal and lifelong thing. From the first consciousness of the child to the clouded darkening of the dying man's mind it goes on. It would go on, in one way or another, if there were no schools, but there are many urgent factors that call for the best effort possible to teach our young. There are the indescribable riches of the world without and within, the inexhaustible store of past human thought and experience, the turbulent present with its significant experiences, the swift passage of a human life and the long tomorrow of eternity.

—*John E. Riley*

The Doctor Goes to Court

Joseph Cohen, LL.B.

Kansas City, Kansas

So you have been asked to be an expert medical witness. If you are experienced in medical legal testimony, then what I have to say tonight may be old to you. But if you only occasionally grace our courtrooms as expert witnesses, I may then have in the lecture to follow a great deal of information which may prove valuable. On the other hand, even if you are an experienced witness, you may find a number of things in this paper which will help to explain the examination and cross-examination, the objections thereto, and the drama that is part of every trial.

I firmly believe that one of the reasons so many physicians and surgeons shy away from the courtroom, aside from the question of economics involved, is that they do not fully understand and appreciate the role of the medical expert in litigation. After they have apparently given a forthright chronological report of the history, the chief complaints, the clinical examination, the x-ray examination, the treatment given to the patient, the diagnosis, and prognosis, some physicians consider it not only annoying and aggravating but a show of unnecessary hostility when the attorney on the other side of the case undertakes to explore the truth and the reasonableness of that testimony by means of a vigorous, searching, and minute cross-examination.

If we understand at the outset that the principal purpose of litigation is to arrive at the truth, and that counsel on each side of the case is trying as skillfully as he knows how to present the facts to the judge or jury in the light most favorable to his client, then you will have no difficulty in understanding the judicial courtroom process.

If you find the lawyers difficult at times, remember that they, too, are sometimes baffled at the difference between the hospital room statements of the medical profession and the courtroom statements which they hear from the witness stand. It is sometimes hard to understand why a patient will be prescribed a course of treatment for an ailment by the doctor who will be heard to testify for the defense that there is nothing wrong with the claimant because that doctor can find no objective evidence of disability. Why, wonders the attorney, will he prescribe massage and physiotherapy for an ailing back and then take the witness stand later on and testify that there was nothing wrong with the patient?

As we enter the atomic age with the complexities of our modern civilization, our highly mechanized industries, speeding motor cars, and jet propelled planes, we encounter a greatly increased number of cases involving personal injuries. Thus, the role of the physician as an expert witness becomes more and more important.

I believe it makes sense when I say that a physician has no right to decline to give his services to a patient involved in litigation and at the same time expect to be favored with that patient's medical business and that of his family and friends when they are not involved in litigation. Leaving out of consideration the fact that members of the medical profession are not immune from compulsory process to bring them into court, a method seldom used for obvious reasons, there is a strong moral obligation on the physician to deal fairly with his patients and with the courts.

Once we break down the confidence of the people in the judicial process, without offering them a better substitute, we tear down the structure of democracy which has given us the highest standard of living and more civil rights and liberties than are enjoyed by any other people on the face of the globe.

The medical profession has a monopoly which cannot be invaded. If we are to keep a free, voluntary society, if we are to stop tendencies toward socialization, if we are to avoid concentrating the medical legal work in the hands of a few, and not always the most competent in the field, it becomes highly important that each man or woman in the medical profession be willing to come to court and testify.

I can say to you men of the medical profession that you are going to find in the future a much better prepared bar in the medical legal field than we have had in the past. Negligence and workmen's compensation cases comprise by far the largest amount of litigation in the courts. Lawyers are learning that they must have specialized skill and training in this field if they are to do a good job on behalf of their clients. Many of us have for years delved into the medical books and journals in the preparation of our cases in order that we might meet on more equal ground the doctors whose opinions we must test in the crucible of litigation.

Now a number of the leading universities are hold-

Although the JOURNAL ordinarily publishes in this section only articles of scientific content, this address seemed to contain sufficient information of value to a large group of physicians to make its publication here advisable. Mr. Cohen presented the paper before the Wyandotte County Medical Society on January 19, 1954.

ing seminars on the graduate level for practicing attorneys, and in connection with the seminars many leading men of the medical profession are giving of their time and talent to the education of the bar. My son who is a second-year student at Harvard Law School tells me that this year the third-year law students go to the Harvard Medical School where members of the faculty lecture to them on medical legal testimony. One of the leading universities in this field is Texas University, and another leader is Tulane. Our own law school at the University of Kansas City also is holding annually a three- to five-day lecture series given by recognized men in the medical profession.

Therefore, I say to you that the younger lawyers are coming out from the law schools better prepared than before, and we older practitioners are trying not to lag behind. I would like to see the University of Kansas School of Medicine make a distinct contribution in this field. If it would sponsor a course for law students and practicing attorneys, there would be reciprocal advantages to the bar and the medical profession and, of course, to the public.

Your function in the judicial process, as medical experts, is to give your best opinions, based upon your education, knowledge, and experience, on the subject matter before the court. Anybody can testify as to what you prescribed and did for the patient, but only you can testify as to why you did it.

Before you are permitted to testify as an expert, counsel offering you as his witness must qualify you. To do so, he inquires as to your educational background, when you received your degree or degrees, when you started to practice, whether you are a specialist or a general practitioner, what professional organizations you belong to, and sometimes whether you have held official positions in those organizations, your postgraduate training, and the states where you are licensed to practice. The purpose of eliciting your qualifications is not then to determine your skill or want of skill nor to parade before the court or jury the vastness of your medical learning, but merely to lay the groundwork or foundation which will permit you to testify as an expert (*A. T. & S. F. R. Co. vs. Sage,* 49 Kan. 524).

Many times you will hear opposing counsel say that he waives the qualification of the witness. Such a waiver may dispense with the necessity of having you testify as to your educational background. On the other hand, opposing counsel may prefer to qualify his expert witness in order to show the court or jury that he has brought into the courtroom a person of unusual training, and the judge will permit him to do so even though the qualification has been waived. If you are a specialist in your field, the lawyer will want to show the extent of your specialized training. Having qualified, you are then permitted to testify what you have seen, heard, or done for the patient, the history, complaints, diagnosis, treatment, and prognosis. You may be asked for your opinion as to the permanent nature of the disability or whether it is of a temporary nature or has been entirely cured. If there is permanent disability, you may be asked in a workmen's compensation case to give your opinion as to the percentage of disability which the injured workman has suffered.

Such a question always deals in the inexact field of opinion. However, for the purpose of the law, you are asked to assume and take into consideration the amount of work which an ordinary, normal, well workman might perform in the general field of labor, and then determine the percentage of loss the injured workman has suffered from his ability to do and perform general manual labor. No one realizes more than I do the great responsibility which such a question poses to every man in the medical profession who is asked to make such a rating.

Our courts have said that a medical expert need not answer questions with certainty. All he need do is give his opinion (*Roark vs. Greene,* 61 Kan. 299).

Your opinion on cross-examination may be tested by opposing counsel in a number of different ways, depending upon his resourcefulness, ingenuity, and skill. You may be asked whether you agree or disagree with certain statements appearing in recognized medical books and authorities, and for that purpose counsel may read to you excerpts from such books. These medical books are not admissible in evidence to establish the declarations or opinions which they contain (*State vs. Baldwin,* 36 Kan. 1). However, the Supreme Court said (*Stout vs. Bowers,* 97 Kan. 33, 36):

"One of the recognized methods of testing the knowledge of an expert witness who founds his opinions on standard medical authorities is to read from those authorities upon the subject in question and interrogate him as to whether his opinions coincide with those expressed in the books and whether there is not a conflict between the opinions he then gives and the views expressed by the authorities upon which he relies for information."

Before you have completed your direct or cross-examination on the witness stand, you may be asked a hypothetical question. Such a question must include only facts which have been established in the evidence directly or by inferences fairly deducible from established facts. Counsel may not assume the existence of matters material to the formation of a correct opinion about which no testimony has been given previously (*Davis vs. Travelers Insurance Company,* 59 Kan. 74; *Western Union vs. Morris,* 67 Kan. 410). However, counsel may base his hypothetical question on weak and inconclusive testimony of one or more witnesses or inferences properly

deducible therefrom (*Roark vs. Greene, supra.*).

It is not necessary that the hypothetical question incorporate all facts supported by the evidence, nor need it include all facts or all theories advanced by the adversary (*New York Life vs. Daerksen,* 75 Fed. 2d 96). Counsel for plaintiff may frame his hypothetical question on facts and theories tending to support his thesis, and he cannot successfully complain if defense counsel in his hypothetical question incorporates only the facts and theories which he desires to support (*Wingfield vs. McClintock,* 85 Kan. 452).

When you are confronted with a hypothetical question, it is well that the physician bear in mind that he is asked to assume the truth of the facts stated therein, and to give his opinion based thereon without interpolating or questioning these facts. It is unimportant so far as that hypothetical question is concerned whether you disagree with the facts or the theory upon which it is predicated. You have a right to rely on the skill of opposing counsel to bring out and stress both sides of the case. Many physicians inexperienced in courtroom tactics find such hypothetical questions difficult and want to argue with counsel about the facts stated therein. Often they have had to be admonished by the court not to do so but to answer the question as propounded.

It has been my experience that the most effective medical expert testimony is given by the physician who is frank and honest and who will concede points to the opposition when they are entitled to such a concession. A recent survey made by one of the legal publications shows that the least effective expert is the one who finds it impossible to concede any point to the other side for fear that he will weaken his own opinion before the court or jury. Our court has said that an expert witness is to be given such credence and weight as the court or jury believes he is entitled to (*Santa Fe vs. Thul,* 32 Kan. 255; *Baird vs. Shaffer,* 101 Kan. 585).

Objections by opposing counsel to a hypothetical question must be specific and must point out to the court specifically in what way the question is deficient. Usually he will point out that it incorporates certain facts not proven, or misstates proven facts, or makes inferences not justified from the established proof. Only such specific objections may be considered by the court. Young, inexperienced lawyers often must learn this rule the hard way after their general objections are overruled by the court (*Linscott vs. Hughbanks,* 140 Kan. 353, 357).

A medical expert need not feel under tension when subjected to cross-examination. Nor should he take offense if counsel tries to weaken the effectiveness of his testimony. Perhaps the soundest and safest attitude is that which men of science should always adopt—that is that you are aiding the triers of the facts to arrive at the truth. While you would be less than human if you did not want to see your opinion prevail in a courtroom, and perhaps you would be lacking in a good sense of justice if you did not seek to persuade a jury or court to adopt your opinion, on the other hand, having given your opinion, you have performed your full duty and you must then leave the advocacy to the lawyers.

X-rays, cardiograms, encephalograms, and other mechanical aids to diagnosis and treatment are common to your profession, but are not so well known by the bar and certainly not well understood by the layman. X-rays and cardiograms have to be taken and interpreted by men with special skill in that field. Therefore, unless your qualifications indicate that you possess such skill, you may not be permitted to testify as to what the x-rays or cardiograms reveal. On the other hand, if you intend to testify concerning x-ray or cardiographic findings, the x-rays and cardiograms must be brought into court, properly identified, and introduced in evidence so that you may be subjected to cross-examination thereon, and in order that opposing counsel might have the films examined by other men in the profession to test your correctness thereon (*Lefebvre vs. Western Coal Co.,* 289 p. 456).

I might say here that these diagnostic aids are so common to your profession now that if you fail to use them in cases treated by you where indicated, you may be subjected to a claim of malpractice, and it will be no defense for you to claim that the patient did not request that you use an x-ray or take a cardiogram. That was the finding of our court (*James vs. Grigsby,* 114 Kan. 627), where the physician was charged with malpractice in the setting of a broken leg without the use of an x-ray.

Hospital records are always important in a law suit. These are admissible in evidence only when the physician, internes, and nurses identify the records as records made by them in the course of the hospitalization of the patient (*Bowman vs. Coyle,* 124 Kan. 492, 495). In order to avoid such a circuitous manner of establishing the veracity of hospital records, counsel many times will agree between themselves that the record may be introduced in evidence without such qualifying proof.

Those who operate hospitals for the benefit of the public must realize that they, too, are part of the judicial system in this democracy, and even though there is some hardship imposed upon hospitals and some expense, too, it is not asking too much to request that they bring, through their librarians and record clerks, these records into court for examination and testimony. As for myself, I try to see to it that the record clerk is reimbursed to the extent of her taxicab fare when she comes to court with the

record, and I am sure that every thoughtful lawyer would consider that a minimum requirement of co-operation on his part.

Your own private records in these cases are also admissible in evidence, and you must be prepared to bring them into court with you for the purpose of refreshing your own recollection about the case, and you must also be prepared to allow opposing counsel to examine records in the hope that he might find therein something contradictory to what you have previously testified.

The fact that you are a member of a busy profession, that of administering to the weaknesses and ills of humanity, does not give you any immunity from compulsory process. But an attorney should and must respect the demands made on your time, should give you notice of the time when you will be needed, and will excuse you from attending at the specified hour if an emergency makes your attendance impossible. On the other hand, your co-operation is needed and should be whole-heartedly given in order that the judicial branch of government might function properly.

There is adequate power in the law to compel you to attend after you have been served with a subpoena, but I assure you that no lawyer would deem it a pleasant task to send the sheriff after an expert witness who adamantly refuses to obey the process served on him. I learned as a young lawyer by bitter experience than an unwilling, unco-operative medical witness can do more harm than good if he is compelled to come to court against his will. Having subpoenaed a doctor to testify in a deposition at Wichita, and being confident that he could testify only one way because he had already given a statement and signed a death certificate as to the cause of the man's death, I insisted that the physician give his testimony.

I probably would have not done so had this physician not insisted on the payment of an unreasonable fee in advance of his testimony. You can imagine how surprised I was when the physician made a complete about face in his testimony, stating that after full consideration of all the facts in the case, he had decided that the death was not due to food poisoning but was due to some other cause. Probably I would have been smarter had I paid the unreasonable fee demanded by this unscrupulous practitioner.

Privileged Communications. Section 60-2805, General Statutes of Kansas, 1949, sets out the various professional groups who are not privileged to testify concerning communications arising in the course of their professional conduct, such as attorneys and clients, priest and parishioner, physician and patient, etc. It states that a physician or surgeon is incompetent to testify concerning any communication made to him by his patient with reference to any physical or supposed physical disease, defects, or injuries, or the time, manner, or circumstances under which the ailment occurred or concerning any knowledge obtained by a personal examination of any patient without the consent of the patient. However, it further provides that if a person, without objection on his part, testifies concerning any such communication, the physician communicated with may also be required to testify on the same subject as though the consent had been given.

That means that you as a physician or surgeon cannot testify as to these communications which come to you in the course of your professional undertaking, but if the patient files a claim or brings a law suit and testifies concerning his condition, then you can be compelled also to testify thereto because the privilege is then waived. After a patient's death, the heirs or personal representatives may waive the objection (*State vs. Pullman,* 85 Kan. 237, 238; *Gorman vs. Hickey,* 145 Kan. 54, 61, 63). On the other hand, if no heir or personal representative of the deceased objects to your testimony, you may testify for the reason that this law is not a privilege given to the physician, but it is a privilege given to the patient (*Doty vs. Ice Company,* 118 Kan. 323; *Kirsch vs. Federal Life Insurance Company,* 149 Kan. 309, 313).

Malpractice. The obligation of a physician and surgeon is merely to use such skill and foresight in the practice of his profession as is generally used by men in the profession. A physician does not impliedly contract to cure his patients (*Tefft vs. Wilcox,* 6 Kan. 46). In case of doubt on which of two courses to follow, all the physician is required to do is to use his best judgment, and he does not guarantee that he will get good results; if he has bad results he is not subject to civil liability (*James vs. Grigsby,* 114 Kan. 627; *Paulich vs. Nipple,* 104 Kan. 801). In one case (*Rainey vs. Smith,* 109 Kan. 692), our court said that an action for malpractice would lie where a wrong diagnosis was made of the plaintiff while she was pregnant. On the other hand, an honest mistake in diagnosis made where you do not treat the patient does not render you liable. In another case (*Bugg vs. Security Benefit Association,* 173 Kan. 522), a physician was sued for damages for having erroneously diagnosed cancer of a breast. He did not perform the operation for the removal of the breast, and our court said he could not be held liable for this woman's damages because the woman had voluntarily had the breast removed by someone else which was the proximate cause of her injury.

However, in another case (*Russell vs. Newman,* 116 Kan. 268), the Supreme Court ordered a new trial where the jury granted the patient only nominal damages for injuries arising when a sponge was left in an incision after removal of a kidney, in spite of the fact that there was testimony that no injury was

suffered by plaintiff as a result thereof. The court said that the patient was entitled to more than just nominal damages for such a mistake.

On the other hand, I want to advise you physicians and surgeons that you cannot collect your fee for services rendered where the claim of malpractice is successfully raised (*Abbott vs. Mayfield,* 8 Kan. App. 387). However, my experience proves that you do not have too much to worry about from this angle in Kansas for the reason that want of skill in treatment, diagnosis, and operation on a patient is a matter which only men of your profession may testify as to in court (*Waddell vs. Wood,* 158 Kan. 469). Only in rare and unusual circumstances is it possible to get any physician to take the witness stand against a brother member of his profession where that question is at issue. It is not the purpose of this paper to delve into the ethics or propriety concerning the screen of protection which the men of your profession throw around each other when your skill is being tested by the judicial process.

If a case can be proven against you, you are liable in civil damages not only for your own mistakes but for the mistakes of the technicians, assistants, and nurses in your employ, but you are not liable for any carelessness or negligence by the nurses, attendants, or assistants in the hospital where your patients are sent. Non-profit hospitals, such as those operated in Kansas City, Kansas, are no longer immune to civil liability for damages arising out of the negligence of their employees (*Nicholson vs. Hospital Association,* 97 Kan. 480), since the decision in *Noel vs. Menninger Foundation,* 175 Kan. 751. Employees themselves may be subjected to claims, but this is seldom done because in most cases the employees are not financially responsible and do not carry insurance against such liability.

After almost 29 years of a busy trial practice, much of it in the field of negligence and workmen's compensation, when I come in contact with and listen to the testimony of many doctors, I have learned to classify them not as honest or dishonest, stupid or brilliant, forthright or inconclusive, but rather as pessimists or optimists. When I give to the men in your profession proper understanding and sympathy, I realize that the classification of pessimists and optimists is about as good a classification as one can find.

The pessimist is the best kind of expert medical witness from the plaintiff's point of view. He will take a rather dreary look at the patient's chances for recovery, find good reason for the complaints which the patient makes of pain and suffering, and agree that the percentage of disability which the plaintiff will suffer will be great. The plaintiff's bar loves these pessimists.

The optimist works for the insurance companies and big corporations. The patient is never as sick as he says he is. His complaints of pain and disability are not sustained by the findings. He believes that if the patient would just forget about his pain and go back to work, he would recover sooner. As an optimist, he sees no reason why the injured patient should not be doing a full day's work or, in any event, he minimizes the percentage of disability which the injured workman or plaintiff has suffered. A few weeks or a few months under the benign treatment of the optimist and the patient is as good as ever, ready to take his place in industry as if he had never been hurt. You can well understand why the insurance companies and corporations love the optimists.

It is not up to me to sermonize on this point. If I told you that I believe that the plaintiff's doctors, or shall I say the pessimists, are the more realistic, you might well conclude that I prefer to believe the patient. As a matter of fact, we are dealing with a human equation wherein we must recognize the fact that various people have different thresholds of pain, some are more persistent than others in overcoming disability, and some may endure physical defects and handicaps without yielding to the temptation to complain.

On the other hand, I am satisfied by my experience that there is an element of neurosis in almost every case of trauma and disease and that the physician must treat the mind as he treats the body to the end that after he has cured a physical defect he does not create a mental aberration.

I am happy to say that I have a healthy respect and admiration for the members of your fine profession in this community, the pessimists and optimists alike, as I have found practically all of you with whom I have come in contact faithful and loyal to your profession and your patients, friendly, and helpful, doing an excellent job in upholding the finest tradition of your great profession.

Temperance and labor are the two best physicians of man: labor sharpens the appetite and temperance prevents him from indulging to excess. —*Rousseau*

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

The controversial health reinsurance issue has come back into prominence, and under conditions that make the whole question about as complicated as it can get. The bill would have the federal government underwrite voluntary health insurance plans if they agree to experiment with risks not usually covered.

Although this measure is a major part of President Eisenhower's health program, it became bogged down in the House Interstate and Foreign Commerce Committee when widespread opposition developed. Then the committee chairman, Rep. Charles E. Wolverton (R.-N.J.), turned to one of his favorite subjects, a plan for federal guarantee of private loans to health facilities for construction and equipment. This bill, however, was not supported by the administration.

In an effort to placate the opposition, Mr. Wolverton offered to eliminate a number of objectionable features from the mortgage guarantee bill. At the same time there were reports that he proposed to merge this bill with the administration-supported reinsurance bill. Meanwhile, Henry J. Kaiser made two special trips to Washington to help out his friend, Mr. Wolverton, by putting his weight behind the mortgage loan idea. That was not surprising, inasmuch as Mr. Kaiser had helped to draw up the bill, which would greatly benefit health centers such as those started on the west coast by the Kaiser Foundation.

Mr. Kaiser, saying he was producing a film to promote the mortgage loan plan, went to the unusual extent of making a direct appeal to Washington news correspondents to write favorable copy about the bill.

While these Wolverton-Kaiser maneuverings were taking place on the mortgage bill, it became apparent that President Eisenhower was not ready to abandon the reinsurance idea. He called a number of executives of major life insurance companies to the White House to try to impress them with the merits of reinsurance and in other ways indicated he still wanted to see the bill passed this session. Secretary Hobby, whose original testimony for reinsurance had been restrained, also joined in the last-minute campaign. But it appeared the tangle might be too complicated even for Mr. Eisenhower to unravel before adjournment.

Most other parts of the Eisenhower health program were moving through Congress, even though some were off schedule. (Of the major bills, A.M.A. opposes only reinsurance.) Legislation to expand the Hill-Burton hospital construction program cleared what might have been a serious obstacle when it was reported out by the Senate committee. Compared with the House bill, the Senate bill gave more discretion to state health authorities in use of funds for constructing facilities for the chronically ill, for nursing homes, and for health centers. However, the Senate would require that funds earmarked for rehabilitation centers be used for the stated purpose. The Senate also would rule out the possibility of U.S. grants to centers devoted solely to treatment. Unless the facility could qualify as a diagnostic center, or a diagnostic-treatment center, it could not be eligible under the Senate bill. This safeguard was not in the House bill.

Of the remaining legislation of interest to the medical profession, the status at this writing was about as follows:

The doctor draft amendment, to strengthen Defense Department's hand in dealing with physicians who might be security risks, had passed the Senate, been reported by the House committee, and was almost a law. Also about to be enacted was a provision liberalizing medical expense deductions from taxable income. The long-dormant bill to transfer responsibility for Indians' health matters from the Indian Bureau in Interior Department to Public Health Service in the Department of Health, Education, and Welfare was pointed toward enactment, but might possibly be held up by objections of Senators from a few western states. The Interior Department had dropped its original objection.

The House-passed social security bill, with the compulsory coverage of physicians eliminated, was before the Senate Finance Committee, where anything could happen. Two bills of medical interest already had been passed by both houses and signed into law. One prohibits the shipment of fireworks into a state where fireworks are illegal, and the other relieves Army medical officers of the technical responsibility for supervising preparation of food.

A reassuring note was sounded by President Eisenhower when he forwarded to Congress the controversial International Labor Organization convention on minimum standards of social security with a recommendation that it not be ratified. His message said most of the points—including a suggestion for socialized medicine—were not proper subjects for the Congress to deal with.

Physicians visiting Chicago are invited to tour the A.M.A. building at any time. Records show that the number of physicians and medical society executives taking the tour during 1953 more than doubled the number for the previous year.

PRESIDENT'S PAGE

DEAR DOCTOR:

During the hot summer many of us look forward to a restful vacation. Some turn to fishing, others relax on the beach, and still others might find a trip to the mountains most restful. While it goes without saying that one is entitled to a certain amount of leisure, it is most heartening to see the program of The Kansas Medical Society go forward.

The month of July is filled with meetings of our various standing committees earnestly working to carry out unfinished business of the preceding year, or introducing new projects in an effort to further medicine professionally and from a public relations standpoint.

It has been necessary to appoint special committees to carry out programs not specifically designated for a standing committee. Your response in this regard has been most gratifying.

The doctors in Reno and surrounding counties who have already had several meetings to discuss and work out a program for our 1955 annual session are holding another such meeting this month.

While your officers are most willing to do what they can to further Society activities, the success of our program is determined by the continued effort of individual members. Committee chairmen will introduce to their committees worth while suggestions from any member of the Society.

May I thank you on behalf of your officers for your excellent co-operation to date and encourage your continued participation in the year ahead.

Yours sincerely,

Murray C. Eddy, M.D.

EDITORIAL COMMENT

Federal Health Programs

PART I

During the fiscal year closing June 30, 1954, the federal government will have appropriated some $1,775,882,197 for health activities. This is more than 1¾ billion dollars, and in the final accounting the figure might even surpass that amount.

According to an analysis prepared by the A.M.A. Washington office, the federal government operates or participates in more than 60 health, medical, and related programs which are operated by 19 different departments, agencies, or commissions. Not all these agencies can give accurate fiscal expenditures until the year is concluded, but the figures are presumed to be reasonably correct.

Some of these programs are operated directly at the federal level, but most are largely distributed to the different states upon some pre-determined formula. How Kansas uses these and other funds will be recorded in future articles. There will be no attempt made to criticize any program mentioned. This series is intended to be explanatory. Statistics cannot be avoided if this story is to be told.

The federal government is in the business of health care. This series of articles will attempt to describe these programs. The physician interested in this subject may then determine for himself whether or not he approves of what is being done.

The Department of Health, Education and Welfare spent a total of $340,553,000 during this fiscal year, or about one-fifth of the total federal health expenditure. All the following are administered under this one department.

The Office of Vocational Rehabilitation used $23,655,500, of which $655,500 represents federal administrative costs. The remainder was given to the states on a dollar for dollar matching basis to give medical care, hospitalization, prosthetic appliances, vocational training, etc., to handicapped persons.

The Food and Drug Administration spent $6,250,000 to enforce laws designed to protect the public against adulteration and the misbranding of foods, drugs, and cosmetics.

The Children's Bureau spent $31,525,000 for crippled children and maternal and child health programs. Federal administration expenses are $1,525,000 so 30 million was spent to match state appropriations for such programs.

The Bureau of Public Assistance spent $50,000,000 for medical and health programs. Federal administration is about $1,550,000. This is a complicated formula, and a complete description would require more space than its value warrants. The briefest account is to say that the federal government spent $1,341,550,000 for public assistance and that about 4 per cent of this total was for health care. This was paid either directly to the vendor or to the client in the four assistance categories—old age, blind, permanently disabled, and dependent children. Included in the above figure is about 50 per cent of the administrative expenses within the various states.

Also included under this major department is the United States Public Health Service which operates all of the following medical programs. These are briefly recorded as follows:

The Office of the Surgeon General spent $2,900,000 for administrative expenses, surveys, international health activities, training programs, etc.

Tuberculosis Control spent $6,000,000, of which something more than two-thirds was given to states for case finding, treatment, and follow-up services on a basis of two federal dollars for each state dollar spent. The remainder was used by the U.S.P.H.S. to finance direct activities including drug evaluations and BCG vaccinations.

Venereal Disease Control spent 5 million dollars, of which just under 2 million was given to the states for treatment and control on a two-to-one basis, the federal grant being twice the state expenditure. Something over 2 million dollars was used for training programs or to pay salaries of federal employees assigned to the states, and the remainder was spent directly by the U.S.P.H.S. for tests and demonstrations.

Assistance to the states accounted for $13,250,000, of which just over 3 million dollars is spent directly by the U.S.P.H.S. for consultant services, training programs, etc. The various states received $10,135,000 on a basis of two federal dollars for each state dollar for general public health purposes.

Communicable Disease Control used 5 million dollars, all of which was spent by the Communicable Disease Center at Atlanta, Georgia, for studies in epidemiology, laboratory services, and special services to the states in case of epidemics.

Engineering, Sanitation, and Industrial Hygiene spent $3,162,500, of which $87,000 was for administrative expenses. Water supply and pollution control used $903,500; studies of radioactive substances $275,000; sanitation, milk and restaurant inspection, garbage disposal, etc., $799,000; health hazards of manufacturing and mining $550,000, and environmental research $547,500.

Alaska received $1,082,000 for public health services and to finance the Arctic Health Research Center at Anchorage.

Hospitals and Medical Care used just over 33 million dollars for the operation of hospitals and health

services for American seamen, Coast Guard and Public Health Service personnel and their dependents, for federal employees injured at work, for leprosy patients, and for narcotic addicts.

The Foreign Quarantine Service spent $2,900,000 to operate 250 quarantine stations located on the borders of the United States. It also operates about 25 medical stations in foreign countries where aliens who seek to enter this country may be examined. And, finally, this service inspects all seagoing vessels and aircraft entering the United States.

The various National Institutes of Health spent $71,153,000, of which the National Cancer Institute received $20,237,000. About two-thirds of this latter was distributed as research grants, just over 2 million dollars was allocated to the states for cancer control programs, and the remainder represents the cost of operating the Bethesda (Maryland) Clinical Center.

The Mental Health Institute received $12,095,000, of which 60 per cent went to individual investigators working on research programs. Two and one-quarter million dollars was allocated to the states, and the rest was spent at the Bethesda Clinical Center.

The National Heart Institute received $15,168,000 and diverted this money according to the same formula as above. Similar also are the Dental Health Institute, $1,740,000; the Microbiological Institute, $5,738,000; the Institute for Neurological Diseases and Blindness, $4,500,000; the Institute of Arthritis and Metabolic Diseases, $7,000,000, and the National Institutes of Health, General Funds, $4,675,000, except the last named fund is almost all used for grants and some of the others do not distribute money for state uses.

Also under the United States Public Health Service is the hospital construction program which is listed under three divisions, obligational cash for new construction under the Hill-Burton Act, 65 million dollars; liquidational cash to complete previously authorized projects, $19,700,000, and administrative expenses $875,000. This program also operates under a complicated formula and with local recommendations, but in general it represents aid for hospital construction whereby the federal government contributes from one-third to two-thirds of the total cost, depending upon the per capita wealth of the state. Since 1946, when this program was started, federal funds have partially financed 2,100 projects with 102,000 hospital beds and in addition aided in the construction of 400 health centers.

This, then, is a summary of health expenditures through the Department of Health, Education and Welfare—some 340 million dollars out of a total of 1¾ billion spent for health by the federal government. The discussion will be continued next month describing such agencies as the Veterans Administration, the State Department, and others.

Malpractice Insurance

Some considerable confusion has arisen over the subject of professional risk insurance for physicians. A generalized national increase in malpractice actions, together with the apparent trend of higher judgments awarded by juries, has caused policy changes among insurance companies.

The majority have organized to agree upon a standard policy, uniform acquisition practices, and equal premium rates. In brief, these companies set rates for each state according to the experience within the area. As may be expected, the cost in coastal and industrial regions is considerably greater than for Kansas. Policy types vary according to specialty practice, and rates differ in certain fields. There are some uniform exclusions such as spinal anesthesia and shock therapy written into all of these policies. The physician is also required to purchase certain other forms of liability protection such as automobile or fire from the same company. However, he may buy practically unlimited protection.

A very few companies did not join this group and continue with their own individual insurance customs. One company issues a policy notably less expensive but operates upon a strict theory of low coverage with no exclusions and careful attention to defense. Both theories have found support in Kansas, and it is not here intended to argue the point. Essential differences should be understood, however, so this primer is prepared to represent an impartial review upon this subject.

The first comment is that every physician should carry some form of insurance protection for each day he is in practice. For the great majority this will never be needed, but its service is not only protection against carelessness but principally to cover suits for situations that are completely beyond the physician's control.

Some members continue their mistaken belief that The Kansas Medical Society provides insurance protection. This is not true and should be understood. The Society elects a Defense Board of three physicians who will assist, if the case appears defensible, any member requesting such aid. The attorney for The Kansas Medical Society may be provided to aid such member, but the cost of his services will be paid by the defendant. The Society will aid in defense if it appears proper, but the Society will not pay judgments or court costs or attorney fees arising out of any malpractice suit.

Second, the individual physician should determine

for himself how much insurance he wishes to have and what company he proposes to select for this protection. Physicians who are entering practice might profit from consulting with established physicians regarding local custom and experience. Philosophy upon this subject will vary. Some will want high limits to protect their personal property against loss by court action. Others will argue that careful defense and low limits will serve to reduce the size of judgments. This decision should be based upon personal preference.

One theory holds that high limits invite suits and subsequent high judgments. The other declares low limits to leave the doctor vulnerable to personal loss. One holds that careful attention to details and thorough defense preparation practically eliminate this danger and argues that experience proves this to be true. The other theory simply denies that statement and speaks of hazards that accompany less than full financial security. One says they have proved the soundness of their position, the other asks what about tomorrow. Low limit advocates tend to credit those holding the other theory with a considerable responsibility for the present situation, while they in turn argue that the present situation presents additional proof for the need of carrying high limit protection.

One theory holds that the plaintiff is more interested in suing the impersonal insurance company than the doctor, but that also is argued against. High judgments and liberal out-of-court settlements are contagious. One such action frequently results in other cases being filed from the area, but again the argument can be turned both ways. So, again, this discussion is not intended for advice on the question of amount. The important thing is that the physician carry insurance; how much and with which company shall be decided by himself.

Regardless of that choice, there are many things a physician may do to prevent his becoming involved in legal action of this type. Even though all may be summarized by saying that careful attention to the practice of sound, scientific medicine and conscientious adherence to the principles of ethics represent the best possible protection, perhaps a few points should still be mentioned. The JOURNAL is indebted to the Medical Protective Company of Fort Wayne, Indiana, for permission to reprint this list of 30 suggestions which every doctor should follow.

1. Keep accurate records permanently. Suits sometimes arise scores of years after the rendering of services.

2. Keep abreast with the average standards of his field of practice in his locality. This the Law requires him to observe.

3. Avoid criticism of the work of other doctors. To

SERVICE SEPARATIONS

As a service to physicians and communities in this state desiring additional medical personnel, the Journal of the Kansas Medical Society will publish in this column each month the names of medical officers who will shortly be separated from the armed forces. These are men who volunteered from Kansas, and many of them will probably be interested in finding locations in this state. Anyone interested in contacting these physicians may write to the address here given.

Theodore L. Batchelder, M.D.
Garden City, Kansas

Albert I. Decker, Jr., M.D.
826 Alabama Street
Lawrence, Kansas

Jack A. Dunagin, M.D.
1266 Collins
Topeka, Kansas

Lyle Harris Edelblute, M.D.
1210 Thurston Street
Manhattan, Kansas

Edgar F. Ewen, M.D.
1022 East 5th
Cherryvale, Kansas

Simon E. Hershorn, M.D.
Wesley Hospital
Wichita, Kansas

Everett E. Howard, M.D.
1708 North Grand
Pittsburg, Kansas

J. D. Kabler, M.D.
1506 Parker Avenue
Wichita, Kansas

J. Gilleran Kendrick, M.D.
4224 East English
Wichita, Kansas

Francis X. Lenski, Jr., M.D.
703 South Cottonwood
Iola, Kansas

Stanley R. McEwen, M.D.
447 North Belmont
Wichita, Kansas

Alexander H. Milne, M.D.
2420 West 25th Street
Topeka, Kansas

Owen C. Peck, M.D.
2000 Oakland
Kansas City, Kansas

Ralph B. Reauwe, M.D.
524 West 7th
Junction City, Kansas

Julian W. Reed, M.D.
703 North Pine
Beloit, Kansas

Robert N. Shears, M.D.
15 Carlton Road
Hutchinson, Kansas

Grant M. Stevens, M.D.
305 Crescent Boulevard
Hutchinson, Kansas

Charles E. Wilder, M.D.
Winter VA Hospital
Topeka, Kansas

do otherwise not only invites retaliation but also weakens his own defenses.

4. Avoid admissions against his own interest on services he himself has rendered or anyone else has rendered in his behalf. Any such admissions will probably be used against him.

5. Learn and observe his legal duty to his patient.

6. Use the x-ray as a diagnostic or confirmatory aid where that is established practice.

7. Guarantee no results. Such guarantees are not only unethical but uninsurable.

8. Co-operate with the patient in securing aid of consultants. These strengthen defenses.

9. Give proper notice of termination of services so as to avoid charges of abandonment.

10. Secure proper written consent for operation in every instance. Without such evidence he may be charged with assault.

11. Keep his relations with his patient strictly confidential.

12. Avoid divulging that he has professional liability coverage.

13. Refrain from unapproved or experimental procedures.

14. Be courteous and tactful in relations with patient.

15. Advise patient of any absence from practice and recommend or make available a competent substitute.

16. Make certain that proper instructions are given with prescriptions or in the care of the patient.

17. Secure proper authorization for any autopsy in which he participates.

18. Have, whenever possible, unprejudiced witnesses of his relations with the patient, especially in the case of female patients.

19. Have a clear understanding relative to his fees before services are rendered, especially if such fees are of sizable amounts.

20. Postpone, whenever possible, any suit for fees until after the statute of limitations on malpractice suits has expired.

21. Secure legal advice before attending a coroner's inquest as a witness.

22. Secure legal advice on how to make his personal assets less vulnerable to damage suit attacks.

23. Avoid sterilization procedures except where medically indicated.

24. Avoid telephonic prescriptions without proper verification.

25. Keep all equipment in proper working order.

26. Avoid fields in which he is not properly qualified.

27. Keep all promised appointments.

28. Give proper instructions to assistants and make certain that they are observed.

29. Maintain due frequency of visits for proper care of patient.

30. Exercise due care at all times, especially in the labelling of bottles or other containers, in the use of mouth gags, in the close observation of casts, in periodic check of needles and other equipment, in requiring personal examination for commitment proceedings, and similar procedures.

Physician Placement

Kansas physicians are aware of the success of the Kansas Plan in helping place doctors in communities where medical care is urgently needed. At present, 68 communities in Kansas with populations under 500 have medical doctors. More than 150 towns with populations under 1,000 have physicians in their communities.

The work has not been completed, however, and the Executive Office is continuing its efforts to advise inquiring physicians where such situations exist.

It becomes necessary periodically to revise the locations list as various communities obtain the services of a physician and others are placed on the critical list. In some instances a serious situation may exist that is not known to the Executive Office, or the reverse may be true when a doctor locates in a community and the office is not informed of that fact.

In order to revise the list at this time, the help of every physician is needed to inform the Executive Office of places requiring the services of additional medical personnel. If the list is kept accurately, the Kansas Plan will continue to produce results and will help Kansas retain its outstanding record among the 48 states in this endeavor.

Study on Ulcers

Ulcers, caught in time, do not necessarily represent an unusual hazard to longevity, according to a recent release from the Institute of Life Insurance. That fact, along with others, resulted from a study of 15 years' mortality experience among 725,000 life insurance policyholders.

Of about 45,000 cases with a record of ulcers studied by the Society of Actuaries, 96 per cent of them men, those who had been medically treated, without an operation and without having had a hemorrhage, were found to have had a relatively favorable mortality experience during the 15 years covered. Three-fourth of the total ulcer cases were duodenal, the balance being gastric.

Another section of the study, covering persons with gall bladder disorders, indicated that such impairments are much more frequent among women than among men.

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in 2 minutes

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Rapid vasodilating action of Privine relieves nasal congestion in a minute or two—effect lasts for hours.

No interference with ciliary activity or other mucosal function.

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Privine 0.05% Solution in 1-oz. bottles with droppers and in pints.

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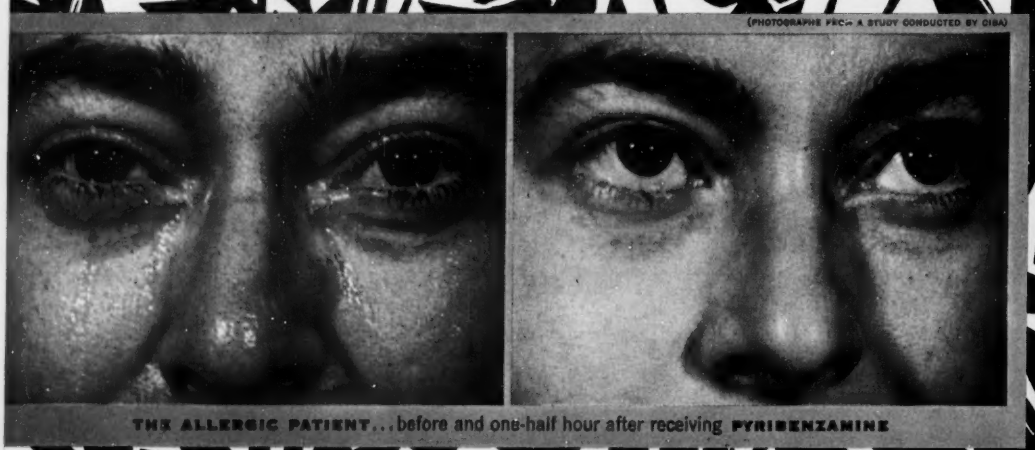
in ragweed hay fever

In the summer and fall of 1953, nine prominent allergists, representing every section of the country except the West Coast, tested Pyribenzamine in a total of 832 patients with ragweed hay fever. The work of these men is significant because of its scope and because it is the most recent major study of antihistamines.
Certain observations are particularly worth noting . . . →



(PHOTOGRAPHS FROM A STUDY CONDUCTED BY CIBA)

THIS ALLERGIC PATIENT . . . before and one-half hour after receiving PYRIBENZAMINE

...of the 832 patients who were given Pyribenzamine, only 84 did not obtain some degree of symptomatic relief.

From this study and from previous investigations involving thousands of allergic patients, one fact is clear: Pyribenzamine gives the allergic patient unsurpassed benefit with antihistamine therapy.

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Try Pyribenzamine – the most prescribed antihistamine – in hay fever, in every allergy susceptible to antihistamine therapy.

Pyribenzamine 25-mg. tablets (coated) and 50-mg. tablets (scored) both available in bottles of 100 and 1000.

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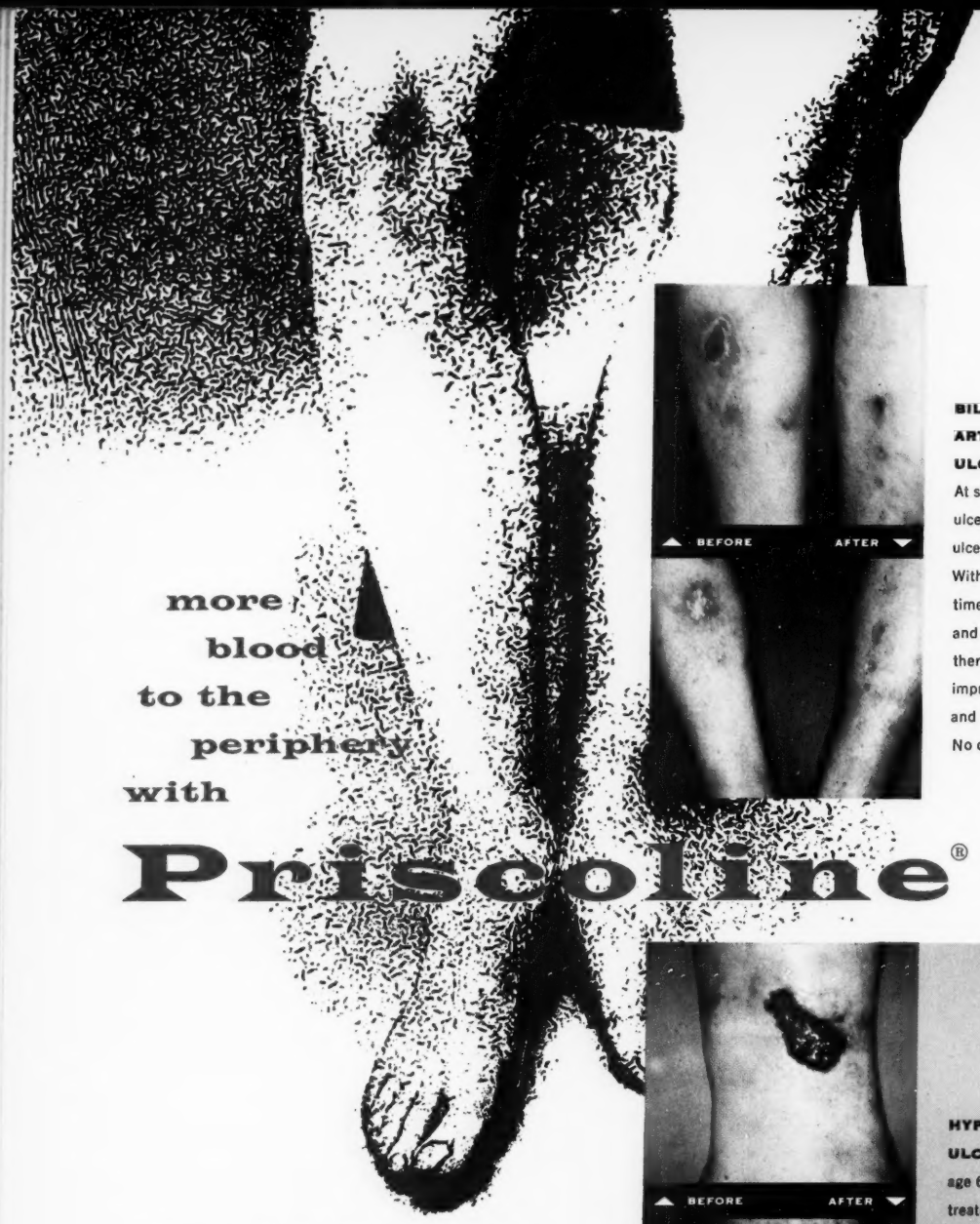

more blood to the periphery with

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BILATERAL ARTERIOSCLEROTIC ULCERATION in patient age 65. At start of Priscoline therapy; ulcer, right leg, 1¾" x 1¼"; ulcer, left leg, ½" x ½". With oral Priscoline, 25 mg. four times daily for one week and 25 mg. every three hours thereafter, there was marked improvement in 2 weeks and healing within 6 weeks. No other medication given.

HYPERTENSIVE ISCHEMIC ULCER of right leg in patient age 65. Ulceration refractory to treatment for 9 months, with patient complaining of severe pain. Treated with oral Priscoline, 50 mg. four times daily for four days and 50 mg. every four hours thereafter. Healing began with onset of Priscoline therapy and was complete in 10 weeks.

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PHOTOGRAPHS AND CLINICAL DATA BY COURTESY OF R. I. LOWENBERG, M.D., CONSULTANT IN VASCULAR SURGERY, CONNECTICUT STATE HOSPITAL, MIDDLETOWN, CONNECTICUT.

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The Complications of Polycythemia Vera

Curtis A. Nystrom, M.D.
Kansas City, Missouri

Polycythemia vera, first described by Vaquez in 1892 and clearly defined by Osler in 1903, has presented several puzzling features to medical investigators.

Polycythemia vera is a comparatively rare disease, characterized by a great increase in circulating blood of the red blood cells and hemoglobin with a color index below unity, hematocrit elevated to 70 per cent and total blood volume increased. In present nomenclature it is called polycythemia hypervolemia.[1] Synonyms are erythremia, splenomegalic polycythemia, Vasquez's disease, Osler's disease, polycythemia rubra, polycythemia with chronic cyanosis, myelopathic polycythemia (Weber), erythrocytosis megalosplenica (Senator), and cryptogenic polycythemia (R. C. Cabot).

The etiology is unknown, but it frequently develops between the ages of 40 and 50, although it occurs at all ages. Records of 6 hospitals indicate that it is higher in Jews of eastern European descent.[4] (Forty-eight per cent of 134 cases—admission rate when average in these hospitals of this race was 10 per cent.) Meakins believes that there is no sex predominance, but Sturgis lists the ratio in males to females as 2:1. Hypotheses as to the etiology include anoxia of the bone marrow,[22, 16, 28] fibrotic narrowing of the vessels,[9, 12] neoplastic changes in the stem cells of the bone marrow,[15, 22, 27] hypersecretion of the intrinsic erythropoietic factor,[21] and hypersensitivity of the carotid sinus and the aortic nerves.[3]

Sodeman[29] feels that anoxia is the basis of polycythemia vera, but he does not feel that every type of polycythemia vera is due to the same type of anoxia. The type described as being associated with hypertension without splenomegaly is caused by stagnant anoxia. Stagnant anoxia is caused by either structural alteration or vasoconstrictor influences. Patients with "splenomegalic polycythemia vera" have a polycythemia which may be viewed as secondary to an underlying process often leukemic or leukemoid in character. This type is due to a competitive anoxia. Initially, competition for available oxygen by numerous immature white cells or later progressive fibrosis and osteosclerosis of the bone marrow with consequent local disturbances to blood flow may result in local anoxia with consequent stimulation of erythropoiesis. This, Sodeman feels, will even account for secondary polycythemias so that the physiologic mechanism is anoxia of the erythropoietic cells. It is erroneously regarded as a primary neoplastic disease of the red cell only because of its frequent and close association with such processes in leukemia and especially in so-called non-leukemic myelosis.

The polycythemic phase may be accompanied by leukocythemia or thrombocythemia or both. Any and all combinations are possible. When it is realized that exhaustion of one element may occur while another begins or continues to hyperfunction, one can more readily understand the wide variation in hematologic pictures presented during the slow progress of this disease. It should be remembered that after therapy some patients have normal values for a number of years, and then the red blood count begins to return to its former high reading, but the hemoglobin may or may not increase. This will cause the stained smear and the color index to present the picture of severe hypochromic anemia.

Diagnosis is generally made by the following criteria:[4] (1) polycythemia which was distinct and persistent and which returned after cessation of therapy, (2) splenomegaly, (3) typical red cyanotic appearance of patient, and (4) absence of any other distinct lesion, such as pulmonary or cardiac, to explain the polycythemia. Other signs and symptoms are headache, vertigo, fainting, and fullness in the head, and in the fundi the veins are much engorged and hemorrhages are frequently seen. There may be disturbed vision and sometimes hemianopsia or diplopia.

Analogous to these symptoms there may be paresthesias and peripheral paralyses. These are usually accounted for by focal hemorrhages or thrombosis in the central nervous system. Other symptoms of central nervous system origin may be periods of slow cerebration, amnesia, irritability, and tinnitus. There is often weakness.

Abdominal pain and cramps are complained of, the former particularly over the splenic enlargement, which may be accounted for by splenic infarctions, and the latter by gastrointestinal infarctions which may lead to hematemesis and melena.[10, 26] Precordial pain indicates myocardial infarction, which is also suggested by the electrocardiogram. The most important signs are cyanosis, a plethoric facies, sple-

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This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school.

Thesis written while the author was a fourth year student. Dr. Nystrom is now interning at St. Mary's Hospital, Kansas City, Missouri.

nomegaly—sometimes assuming enormous proportions. There may be hypercalcemia and a hypochloremia. The basal metabolism rate is elevated by 15-25 per cent or higher. Blood pressure is elevated, and hypertension might be present.

The red blood count is elevated to 7-12 million with a hemoglobin of 150-160 per cent. The color index is below unity. There is possible irregularity in the red blood cells in shape and size; there is always evidence of immaturity as shown by polychromatophilia and normoblastic forms. Usually a leukocytosis is due to increase in granulocytes, but abnormal varieties are seldom found. The platelets are increased.[1] In about 75 per cent of the cases the spleen is enlarged; the liver is enlarged in about 50 per cent. X-ray reveals increased vascular markings in the lung fields. There is frequent bleeding from the nose and gums; this is not due to a tendency to bleeding but is the result of congestion of the vascular bed.[26] Total blood volume is increased.[5, 9]

There are a number of complications listed that occur in polycythemia vera:

Cerebral thrombosis	16.5%
Suspected brain tumor	4.9%
Choked disk	2.4%
Herpes	0.6%
Combined sclerosis	0.6%
Gout (possibly a complication of a pre-existing condition)[6]	4.7%

Strobel classifies polycythemia vera into types according to the degree of leukocytic reaction in the peripheral blood, as follows:[28]

Type I—Simple polycythemia vera, characterized by normal or slightly increased leukocyte count or by a slight increase in the per cent of polymorphonuclear neutrophils.

Type II—Polycythemia vera with mature leukocytosis (more than 15,000 mature leukocytes per cubic millimeter of blood).

Type III—Leukemoid polycythemia vera, characterized by leukocytosis and the presence of immature leukocytes.

(Transition occurs from one type to another in time and following treatment.)

	82 pts. Type I	30 pts. Type II	30 pts. Type III
No change	75%		10%
To Type I			43.3%
To Type II			
To Type III	24.4%	23.3%	20%
Acute leukemia	no-breakdown		

All patients had a five-year follow-up, but 25 of Type I had a 10-year follow-up.

Using this classification, it was seen that splenomegaly and hyperuricemia was about equal in Type I and Type II but was marked in Type III. Gout occurred almost twice as frequently in Type II as in Type I. The serum uric acid was increased in 54 per cent of Type III with 12 of the 14 cases having clinical gout.

Complications which have their manifestation on the skin include hemorrhages with resulting brownish pigmentation. There may be purpuric spots and even hematomata. Eczema and dryness are not unusual. Acne-form, urticarial eruption with rose-red coloration over firm, irregular round or oval nodules, very tender to touch and scattered over the trunk, have been reported.[8]

The appearance of the nail-fold capillaries as compared with normal has led Brown and Giffin[9] to classify vascular changes in polycythemia vera as chiefly concerned with (1) readjustment of the vascular bed to the augmented blood volume; (2) changes in vessel walls, probably due to strain, metabolic disturbances, and various other factors; (3) vascular plugging, probably due to thrombotic processes as a result of increased viscosity and changes in vessel wall and the coagulation factors of the blood; and (4) changes in the erythrogenic tissue.

In the first stages of the disease the accommodation of the blood vessels occurs in the large veins and in the more vascular organs, especially the spleen. As the condition progresses the smaller veins participate in the engorgement, as can be observed in the peripheral and the retinal veins. With more advancement comes the plethoric facies and the bluish-red mucous membranes. Probably the symptoms of polycythemia vera are not produced until the engorgement progresses to the smallest vessels.

Cardiac hypertrophy is often seen following increased pulmonary congestion and increased viscosity of the blood. Vascular diseases include venous thrombosis, varicosities, and phlebitis.[8] Many patients have episodes of phlebitis years prior to development of other symptoms of polycythemia vera.[22]

Vascular symptoms which occur early in patients who later go on to develop or have early polycythemia vera are due to vascular occlusions in the brain, in the coronary arteries, hepatic veins and arteries, and veins of the extremities. There are also circulatory disturbances without clinical evidence of demonstrable occlusion. Since a circulatory disturbance in the limbs, especially the lower limbs, is often one of the earliest subjective symptoms of polycythemia vera and presents much the same picture as thromboangiitis obliterans or arteriosclerotic occlusion of the vessels of the legs, all patients with obscure vascular disturbances should be examined for possible polycythemia vera.[12]

There are vasomotor disturbances of the dilator type suggesting erythromelagia, with features like those of Raynaud's disease. This is possibly explained by the microscopic studies done on the blood vessels

in normal patients, in patients with polycythemia vera, and in those with thromboangiitis obliterans. It was found that the polycythemia vera and thromboangiitis obliteran sections were much alike and showed thickening of the capillary walls with increase in fibrous tissue. They showed marked subintimal and adventitial fibrosis of the arteries and arterioles. Some of the fibrosis extended into and through the media.[4]

Failure to keep the nervous system manifestation of polycythemia vera in mind will lead to diagnostic mistakes. Many of these patients have symptoms referable to the nervous system which may be extensive and sometimes confused with primary neurological conditions or surgically relievable tumors or masses.[7, 12] One must not forget that many diseases are associated with polycythemia vera, and these include those of neurologic nature. A list of many neurologic symptoms and their frequency follows:[7]

Total cases	56
Headache	33
Vertigo	30
General weakness	15
Easy fatigability	13
Fullness in the head Paresthesia Mental impairment	11
Visual disturbances Pain Nausea and emesis	9
Nervousness and irritability	8
Emotionalism	5
Sleep disturbances General pruritus Tinnitus	4

Various symptoms observed in reference to the eye are thought to be caused either by engorgement or by small thromboses. Changes in the fundus are more or less those called cyanosis retinae by Liebreich. The color of the fundus is darker red, the veins are greatly enlarged and tortuous. Many veins are visible because even the smallest veins are filled with blood. Two patients were directly observed to have spasm of the central retinal artery. The veins became greatly dilated and tortuous. The arteries were so free of blood that they appeared as broad white stripes. During these and other possibly similar episodes, the patient's vision deteriorated rapidly until generally only handwaving was visible. With recovery from the spasm, blood could be seen flowing back into the arteries with a resultant gradual, but not complete, return of vision. There was also an example of the formation of new blood vessels and retinal hemorrhages.[13] Meniere's syndrome has been observed and is believed to be caused by distension and engorgement of the vessels of the middle ear.[8] Papilledema is seen as a complication.[16]

Vascular lesions of the brain are the most serious complication of this disease and are a common cause of death. Nervous symptoms are apprehension, nervousness, excitability, irritability, hypochondriasis, disturbed mentality, insanity, delirium, insomnia, muscular atrophy, numbness, epileptiform attacks, muscular twitchings, shivering, tremor, loss of consciousness, aphasia, paralysis, and choreiform movements.[14] Pollack[14] had a case which he thought was due to a lesion in the basal ganglion. This was thought to be due to a definite pathological condition in the brain such as hemorrhage or thrombosis, for choreiform movements later disappeared, but erythremia persisted. (Some authors prefer to use the term erythremia to polycythemia vera.) Any pathological condition in the brain caused by hemorrhage or thrombosis can be produced by polycythemia vera.

Disturbed circulation and abdominal plethora cause many symptoms in the abdomen, some of which must be thought of in an acute abdomen.[11] The patient may have a fullness of the stomach, thirst, gas pains, belching, and chronic constipation. There is frequently pain and tenderness over the spleen, occasionally pain into the left shoulder or down the left side of the body and into the left leg. Hemorrhage, often massive, is an important complication. Thrombosis, when in the mesenteric veins and arteries, may be mistaken for peritonitis.

There are many cases of erythremia listed in the literature. A typical patient entered the Massachusetts General Hospital with chief complaints of dyspnea on exertion, edema, abdominal cramps, and melena. She had had an episode of left leg swelling and pain 17 years before admission. She was admitted to the hospital at a prior time following an acute myocardial infection with acute pulmonary edema and lower abdominal cramps. The patient was treated for her myocardial infarction at that time and was dismissed. The second admission was two years later, and at this time she entered with massive pleural effusion, ascites, pulmonary edema, right- and left-sided failure, hypotension, nausea, and vomiting. The melena was due to mesenteric artery thrombosis.[18]

Friedman finds that the blood of patients with gastric ulcer is generally anemic and with duodenal ulcer it is polycythemic and rarely anemic.[20] He believes that the ulcers found in polycythemia vera patients are due to adrenalin,[19] but the occurrence of thrombosis of a gastric vessel is suggested. The occurrence was 8 per cent in 143 cases, and the incidence of ulcer in 200 controls was 2 per cent.[21]

A 69-year-old female entered the Massachusetts General Hospital with the chief complaint of an abdominal mass which had been increasing in size for seven years. The mass when first detected was the size of a walnut, located in the left lower quadrant and without associated symptoms. Three years prior to admission it had markedly increased in size. The patient now had increased urinary frequency, nocturia, and anorexia. At the same time she began to

notice a tendency to bruise easily. She had some discomfort when erect and required medication to sleep. With increase in the mass size, she had developed moderate dyspnea and some palpitation on exertion.

She had lost 30 pounds in the last three years without nausea, vomiting, or change in bowel habits. No vaginal bleeding occurred since menopause at age of 53.

Physical examination revealed an emaciated female with bronze pigmentation of exposed skin areas and multiple ecchymotic spots over the arms and upper trunk. Her color was high but not violaceous. There were a few telangiectases present over both cheeks and right ear. The heart was enlarged to percussion, and there were irregularly frequent extrasystoles. The second aortic sound was louder than the second pulmonic sound. The mass filled the abdomen except the right upper quadrant. It was not easily movable, but it was not attached to the abdominal wall. A definite notch was palpable along its right border below the level of the umbilicus. It was not palpable rectally, and pelvic examination was within normal limits. The liver was down four fingerbreadths below the costal margin. Distention of superficial veins in the neck and left leg was seen.

Laboratory work revealed a white blood count of 23,850 with 87 per cent neutrophils. Granulocytes showed a shift to the right. The red blood count was 8.26 million with hemoglobin of 18.5 grams. The hematocrit was 76 per cent. The red blood count showed considerable variation in size and shape with some achromia, many cells showing polychromatophilia. Reticulocyte count was 7 per cent with occasional normoblasts. Platelets were diminished, and bleeding time was 1.5 minutes while clotting time was 11 minutes. Stool guaiac examination was negative. Hepatogram revealed some hepatic impairment. A barium enema showed downward displacement of the major part of the colon by an extrinsic mass. Basal metabolism rate on two occasions was plus 69 and plus 59.

On the seventh hospital day a sternal bone-marrow aspiration was done. The myeloid-erythroid ratio was 9:1 with a marked shift of myeloid maturation to the right. No blast forms were seen, and there were rare premyelocytes. Megakaryocytes were plentiful. No plasma cells were seen.

Following a sternal bone-marrow biopsy one week later, which revealed much the same blood picture in the bone-marrow, the patient developed a painful subpectoral hematoma in the site of the biopsy. This was surgically evacuated following clotting, but she had an exacerbation three days later and died two days following this second episode.

It was thought necessary in this case to rule out ovarian tumor since the mass was first noticed in the left lower quadrant, but without vaginal bleeding and with a normal pelvic examination this was not thought likely. Leukemia was thought of as she had such a high basal metabolism rate, but all of the white blood cells were mature. Few patients live this long with the disease, so it is not known whether or not the basal metabolism rate is normal. It could be accounted for by the large amount of erythropoietic tissue found. X-ray showed multiple vertebral fractures involving the lower thoracic and upper lumbar segments, also a rib fracture on the right side, with all bones decalcified (normal at this age). Her thin bones were also due to the fact that they were full of actively regenerating blood, and the trabeculae had been hollowed out by this long, slow disease. At autopsy there was also found blockage of some of the pyramids of the kidneys by urates.[23]

While hepatic enlargement is common in polycythemia vera, the presence of marked enlargement frequently signifies an important complication. Phenlyhydrazine jaundice, leukemic transformation, and myocardial decompensation are not uncommon causes of considerable hepatic enlargement and are readily detectable. Cirrhosis of the liver and thrombosis of the hepatic and portal veins remain to explain a small number of cases, and the condition is usually diagnosed at autopsy.[23]

Study of the collected reports of cases discloses such points of diagnosis usefulness as sudden enlargement of the liver, rapid accumulation of ascites, resistance of ascites to mercurial diuretics, frequent terminal jaundice, and a marked abnormal plasma cholesterol partition in thrombosis of the hepatic veins, occasional decrease in the size of the liver and effective diuretic measures in ascites of cirrhosis, and the infrequency of ascites, hepatic enlargement, and jaundice in the thrombosis of the portal vein.[23, 24]

A basic change in all five cases studied by Newman, Feltman, and Devlin was an increase in the maximum breathing capacity after phlebotomy. This is believed to be due to an increase in the elasticity and a decrease in the viscous resistance of the lung tissue to changes in shape, and, when present, to a decrease in the anoxemia. The increase in maximum breathing capacity was probably due primarily to a decrease in blood viscosity and to some extent a decrease in total blood volume. Three of their patients revealed papilledema, and one had painless gangrenous changes at the tips of the distal phalanges of the four medial fingers of the left hand.[16]

Barker[17] feels that cases in which both polycythemia and chronic pulmonary disease are present should be divided into four groups:

Group I—Primary pulmonary disease with compensatory polycythemia—here blood volume is not increased, and the treatment of the polycythemia would be detrimental to the patient.

Group II—Primary pulmonary disease in which

the polycythemic response has overcompensated and there is an increased total blood volume.

Group III—Polycythemia vera with complicating but unassociated pulmonary disease.

Group IV—Those cases of polycythemia vera causing or predisposing to pulmonary disease. Patients with polycythemia vera have dypsnea on exertion, vital capacity is reduced, and there is pulmonary congestion. These patients are easily affected with an upper respiratory infection. This causes a vicious cycle. The last three groups would benefit from treatment of the polycythemia. Hoarseness is not unusual, for the vocal cords are excessively red.[8]

The only effect that polycythemia vera has on the kidney is that of hemorrhage in the capsule,[25] accumulation of urates in the pyramids,[22] and displacement of the kidney with angulation of the ureter by the enlarging spleen.[10]

There is excessive nucleoprotein breakdown with excretion of considerable amounts of porphyrins[2] and uric acid in the course of active erythropoiesis.[10, 11, 22] This high concentration of uric acids either causes gout or is the overload which brings out an impending case.[29]

When polycythemia vera goes on for years, the condition terminates in many other conditions, such as the subsequent development of myeloma. This is thought to be due to neoplastic changes in the stem cells of the bone marrow. In these cases, the stem cell may first have differentiated into the red cell series to cause the elevated red blood count which later decreased when the stem cell differentiated into myeloma cells.[15] The course is an irregular one, and death is usually due to vascular accidents or a failing circulation. Great enlargement of the spleen may cause left-sided hydronephrosis which may become infected and cause the patient to die of sepsis.[1]

Clinical and hematological changes which appear in the subsequent and terminal stages of the disease are unusual polynucleosis, leukemia, thrombocythemia, chlorotic polycythemia, rare anemia, and their combinations. One must not forget that patients can have the disease and not have any subjective symptoms.

It may be generally stated that those patients who have had the disease for a long time and who do not succumb to a vascular complication or later in the erythroleukemic phase, will ultimately reach marked anemia, and in addition there will be leukopenia and thrombopenia. However, the platelets may persist in large numbers, and anemia may then be associated with thrombocythemia.[26] It was found that in 3 cases out of 15, after having polycythemia vera for many years, there was increasing splenomegaly, anemia, and a leukemic blood picture. In one case examined postmortem, the spleen, liver, and bone-marrow showed a hyperplastic blood formation very similar to that seen in myelogenous leukemia.[27]

Minot and Buckman[27] feel that myelogenous leukemia and erythremia appear initimately related, and both may be of a neoplastic nature. There occur cases of erythremia and leukemia that illustrate multiple varieties of varying degrees of primary pathological activity of the myeloid tissues which usually conforms to a definite type. The development of marked anemia in erythremia, as in leukemia, is dependent upon a disorderly proliferation of the myeloid tissue.

Treatment will not be discussed other than to say that since the etiology of the disease is not known, all treatments are symptomatic. The latest and seemingly most effective therapy is with P^{32}.

COMMENT

Polycythemia vera is a rare disease and is often thought to be confined to the blood picture of high red blood cell count, high white cell count, increased hemoglobin, elevated hematocrit, and increased blood volume, with its resultant congestion in the vessels. The patient generally thought of has a red cyanotic appearance, dyspnea on exertion, and splenomegaly.

This picture of the polycythemia vera patient must be expanded to include all of the symptoms, signs, and complications which can be caused by congestion in the lungs, overloading of the heart with resultant chronic passive congestion of the liver, right-sided heart failure, and storage of red blood cells in the spleen. Congestion and increased viscosity of the blood with its retarded flow in the extremities causes peripheral circulatory failure. When one considers the large range of possibilities which may be caused by thrombosis or hemorrhage in the skin, brain, lungs, heart, liver, spleen, kidney capsule, mesenteric vessels, and hemorrhoids, polycythemia vera and its complications must be ruled out in many patients admitted to the hospital.

We must also think in checking a patient that he might have had polycythemia vera for years and that his present condition may be a terminal phase, or that he has been treated for polycythemia vera and the present state may be due to the continued polycythemia vera and to the effect of therapy.

Finally, we must keep in mind that some patients with polycythemia vera live in good health without symptoms. Others have symptoms which cannot be explained by all of the aspects known about polycythemia vera. Lastly, there can be other conditions associated with polycythemia vera that are either not related to the polycythemia vera or are made worse because of the presence of polycythemia vera.

In summary, many of the various symptoms and signs that have been observed in patients suffering with polycythemia vera have been listed.

Some of the more distinctive complications and some illustrative case histories have been given.

The clinical conditions under which the diagnosis

of polycythemia vera has to be ruled out have been stressed.

A number of complications which polycythemia vera patients develop terminally have been presented.

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Payment of $76,000 an hour—that is the amount spent by all Blue Cross Plans together for hospital service every hour in 1953. This adds up to $1,800,000 every day, or $331,000,000 for a period of six months. This amount represents 91.7 per cent of the total income of all 86 Blue Cross Plans during that period.

ACTIVITIES OF MEMBERS

Dr. Fred J. McEwen, Wichita, addressed the Kansas State Dental Association at its annual session in May. His subject was "Coronary Heart Disease."

Dr. Paul H. Lorhan, professor of surgery and chairman of the anesthesiology section at the University of Kansas Medical Center, recently received a commemorative plaque and honorary diploma from the International University of Mendez Pelayo. The award was in recognition of his services to the practice of anesthesiology in Spain.

Dr. Clarence H. Benage, Pittsburg, was guest speaker at the May meeting of the Crawford County Medical Assistants' Society. He told of advancements in patient care in the past 105 years.

Dr. Henry G. Hurtig, Hanover, was guest of honor at a breakfast given by the Hanover Council of the Knights of Columbus on May 23. The tribute was in recognition of Dr. Hurtig's 50 years of continuous membership and for his many years of service to humanity as a physician.

Dr. W. C. Goodpasture and Dr. George E. Milbank, Wichita, served on the Budget Hearing Committee of the Wichita Community Chest last month.

Dr. Orville R. Clark, Topeka, addressed the Shawnee County Licensed Practical Nurses' Association at its May meeting on the subject of "Need of Improved Nursing Care Due to Advances Made in Surgery in Recent Years."

Dr. M. D. McComas, Jr., Concordia, presented an exhibit on plastic surgery of the kidney at a meeting of the American Urological Association in New York in May.

Dr. Frances Schiltz, Wichita, was presented a certificate of award by the University of Wichita on May 12 for 25 years of service as medical director for women.

Five Wichita physicians took part in career conferences sponsored by high schools in Wichita before

the close of the school year, each speaking on "Medicine as a Career." The five were: Dr. Harold F. O'Donnell, Dr. Edward S. Brinton, Dr. Condon T. Hagan, Dr. Howard C. Clark, and Dr. Dale W. Peters.

Dr. Lester D. Bowles, Clifton, announced last month the closing of his office in Clifton to move to Norton.

Dr. Martin Hagan, Wichita, recently observed the 50th anniversary of his start in the practice of medicine and was the subject of a feature story in the *Medical Bulletin* of Sedgwick County.

Dr. William J. Smith, who has been practicing in Stockton for five years, recently received orders to report for induction into the armed forces on June 30.

A feature story about Dr. and Mrs. Frank A. Eckdall, Emporia, was carried in the *Emporia Gazette* on May 22 on the occasion of their golden wedding anniversary.

Four Wichita physicians were named to office in the Sedgwick County Chapter of the Kansas Academy of General Practice last month: Dr. George Thorpe, president; Dr. Harold Low, president-elect; Dr. Robert Kasha, vice-president, and Dr. Charles McCoy, secretary-treasurer. Serving on the Board of Directors are Dr. Bruce Meeker, Dr. Jay Wisdom, and Dr. Paul Kaelson.

Dr. Yale E. Parkhurst, Belle Plaine, has received orders to report for active duty with the armed forces in July.

Dr. Edward D. Greenwood, of the Menninger Foundation, Topeka, was leader of a study section on "Our Institutional Treatment Program" at a conference on juvenile delinquency held in Washington this month.

Dr. Carter B. Sigel, Cottonwood Falls, will be called to active duty as a lieutenant in the Navy soon. He plans to return to Cottonwood Falls on completion of a two-year tour of duty.

Dr. H. Preston Palmer and Dr. Galen W. Fields, Scott City, announce that Dr. B. Morris Hopkins will be associated with them in practice after July 7. Dr. Hopkins, a graduate of the University of Kansas School of Medicine, has been interning in Washington, D. C., during the past year.

Dr. John H. Hege, who has been medical director at Sunflower Ordance Works for the past 10 months, moved recently to Hartford, Connecticut, to become medical director for the Royal Typewriter Company.

Dr. Michael J. Cox, Dodge City, addressed the Soroptimist Business Women's Club in Dodge City recently on the subject of "Cancer of the Breast." He also showed a film on self examination.

Dr. Ralph L. Hopp, who formerly practiced in Hardtner, has gone to Ann Arbor, Michigan, for a residency. His practice in Hardtner is being taken over by Dr. Marvin H. Cheshire of Memphis, Tennessee.

Dr. Frederick W. O'Donnell, Junction City, was speaker at a formal ceremony held at Fort Riley in June to dedicate Wainwright Hall in honor of the late General Jonathan Wainwright.

Dr. C. C. Hawke recently returned from Europe where he attended meetings of the Association of Surgeons of Great Britain and Ireland, the American College of Surgeons, and the International Society of Surgeons. He gave an account of his trip when he addressed the Winfield Chamber of Commerce at a meeting held last month.

Dr. Dick B. McKee, Pittsburg, was elected president of the Kansas State Board of Health at its annual meeting held last month. Dr. H. St. Clair O'Donnell, Ellsworth, was named vice-president, and Dr. Thomas R. Hood was reappointed executive secretary.

Booklet on Civil Defense

A booklet recently published by the Council on National Emergency Medical Service of the American Medical Association outlines the part the physician should play in civil defense activities. Reprinted from the *Journal of the A.M.A.*, the articles discuss such aspects as organizing for civil defense, developing medical participation in civil defense, the physician's responsibilities, and the doctor and the improvised hospital.

The booklet may be secured on request.

BLUE SHIELD

Prolonged Illness Coverage by Prepayment Plans

It is now recognized by a large number of people that Blue Cross-Blue Shield plans cannot solve community health problems with programs that limit benefits to services rendered only in the hospital. There is probably little doubt that treatment for minor ailments in the home, doctor's office, or outpatient department of the hospital can be paid for more economically out-of-pocket than they can be insured against. This is not true, however, of the services required by persons suffering from protracted illnesses.

It is not economical from a community point of view to maintain a patient in a hospital bed when the illness or injury no longer requires hospitalization but does probably require follow-up care for a long period of time. Let us not place undue emphasis on services rendered outside the hospital, for it is admitted that the basic services rendered in the hospital are the most important part of any prepayment plan. For this reason, it is feasible to offer prolonged illness coverage only to persons who have basic coverage of both Blue Cross and Blue Shield.

Some nine Blue Shield plans have followed the logical development of experimenting with prolonged illness coverage. It is estimated that these nine plans, along with a few commercial companies, now have close to a million members protected against what is commonly called major medical expense.

Most commercial companies in the field operate on the deductible principle, that is, the member pays the first $100 or $200 and the company pays the balance up to $3,000 or $5,000. Some Blue Shield plans have adopted this principle, while others make a maximum payment for specified conditions with no deductible involved.

ILLNESSES LISTED

Illnesses and injuries representative of those covered by prolonged illness contracts are as follows:

1. Poliomyelitis and its late effects.
2. Cancer, including disseminated cancer such as lymphoma, myeloma, leukemia, and Hodgkin's disease.
3. Benign neoplasms of the brain or spinal cord.
4. Subacute combined degeneration of the spinal cord.
5. Cerebral hemorrhage, embolism, or thrombosis.
6. Coronary embolism or thrombosis.
7. Subarachnoid hemorrhage.
8. Rheumatic fever or chorea.
9. Congestive heart failure.
10. Active pulmonary tuberculosis.
11. Ulcerative colitis and regional enteritis.
12. Cirrhosis of the liver with ascites requiring paracentesis or following a shunt operation.
13. Chronic nephrosis or chronic nephritis with uremia.
14. Pemphigus.
15. Myasthenia.
16. Amputations where prostheses are indicated.
17. Fractures.
18. Hemiplegia, paraplegia, or quadriplegia.

Although this list embraces a wide scope of illnesses, it is hoped that more experience in this field will ultimately lead to coverage for any condition requiring care after discharge from the hospital, even if for a short period.

BENEFITS OFFERED

Each member is entitled to benefits for medical, surgical, hospital, and other related services, varying, of course, with each individual plan's contract. It should be mentioned that plans offering this coverage try to let the member assume some responsibility for items that are likely to be used excessively such as drugs, appliances, special nurses' services, and nursing home care. Physicians and visiting nurses are paid in full.

A. Any member being treated for one of the above listed 18 conditions will receive general benefits as follows: (No attempt has been made in this article to list specific benefits since each plan offers different basic coverage and prolonged illness coverage.)

1. Physicians' Service
   a. For hospital visits after expiration of basic Blue Shield coverage.
   b. For medical (non-surgical) services rendered following discharge from hospital.
   c. X-ray, x-ray therapy, pathology examinations, and physical therapy according to fee schedule of contract in force.
2. Room and Board
   a. Number of available days increased to approximately twice that of basic contract.
   b. About 75 per cent of charges, or a daily indemnity for room in licensed nursing home.
3. Medication, Drugs, Appliances and other Ancillary Services
   a. Usually 75 per cent of medications, drugs, and operating room is covered after expiration of basic coverage benefits.
   b. The same applies to drugs requiring prescriptions after discharge from hospital (75 per cent).
   c. X-ray, x-ray therapy, pathology, and out-patient room according to fee schedule.
   d. The entire cost of rental or 75 per cent of

the cost of purchase of appliances ordered by the attending physician.

4. Nursing Services
   a. About 50 per cent of private duty nursing charges up to a maximum of around $250 to $400.

B. Some plans provide most of the above services to members confined to mental hospitals. In addition, benefits are available for electric shock and insulin shock treatments. This applies, of course, only to patients diagnosed as acute mental rather than as custodial patients.

C. In addition to the 18 categories of conditions listed previously, persons suffering from any other prolonged illness *in the hospital* are given an extension of benefits beyond basic coverage. The amount of additional benefits varies from plan to plan.

Perhaps the success of this type coverage is best assured in those plans where 80 per cent or more of their members are covered by a Blue Shield "Service Benefits" clause. Since, in such cases, the charge made by physicians will be that of the Blue Shield fee schedule, and since the basic contract will cover a great part of many of these cases, the prolonged illness rider can be offered at a reasonable rate and still be self sustaining.

D. Kansas Blue Shield is watching these experiments with interest, and it is expected that tangible steps will be taken soon to introduce discussion of the ideas with Blue Shield Relations Committees.

Last year's traffic toll of deaths and injuries was the heaviest in history.

Number of Physicians High

The total number of physicians licensed to practice in the United States, 218,522, set an all-time high record in 1953. Official figures from the 52nd annual report on medical licensure of the American Medical Association's Council on Medical Education and Hospitals indicate that 7,276 persons were added to the medical profession in 1953. During the same period, 3,412 physician deaths were reported.

The report shows that 14,434 medical licenses were issued in 1953 by the examining boards of the 48 states, the District of Columbia, Alaska, Canal Zone, Guam, Hawaii, and Puerto Rico. Of this number, 6,565 were granted by examination and 7,869 by reciprocity or endorsement.

The present high level of medical education in this country is indicated by the fact that of the 5,646 graduates of approved medical schools in the United States to take examinations, only 3.8 per cent failed to pass. In comparison, however, of the 1,463 graduates of foreign medical faculties examined, 45.5 per cent failed.

Of the total number of physicians in the United States at the close of 1953, 156,333 were engaged in private practice, 6,677 were in research and teaching, 29,161 were interns or residents or physicians engaged in hospital administration, 9,311 were retired or not in practice, and 17,040 were in government service.

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Physician's Obligation—Accident Prevention

Physicians, by including advice on accident prevention in their routine health supervision of the child, will increase the parents' knowledge and understanding of child care and management in general.

When giving accident prevention advice, the physician should keep in mind:

1. Hazards at certain ages.
2. Behavior and drives at certain ages.
3. The emotional attitude of the parents toward the child and toward each other.
4. Physical, emotional and intellectual capacities of the child.
5. The physical and emotional environment of the child.

The private physician has the opportunity on home calls to suggest safety measures and to point out hazards. He also can contribute much to help develop community education by working with safety or health groups such as Parent-Teacher Associations and schools.

There are many individuals who believe that when we place proper emphasis upon accident prevention, giving to it the same amount of study, personnel, funds, etc., as we now give to the prevention of communicable disease, we can expect almost comparable results.

Prepared by Committee on Child Welfare

Clinicopathological Conference

CASE PRESENTATION

Dr. Harold Harvey, resident in obstetrics: This patient was a 59-year-old white living female who was a waitress in a tavern. She was first seen in the medicine outpatient department on January 25, 1954, complaining of "swelling in stomach," malaise, eructation, and flatulence of increasing severity for two months duration. The patient began losing weight eight months prior to admission.

The past history revealed the usual childhood diseases with deafness following measles at age three. A "cyst" had been removed from the right side of the neck in 1935 and, apparently, had recurred and increased in size. She had also had an unexplained anemia while working in the powder room of a soap factory in 1932.

The family history was not remarkable other than that one brother died of carcinoma of the liver.

Review of systems revealed the patient to be edentulous, to have a "sore tongue," and a chronic cough with four to five cups of clear white, foamy sputum per day. Menstrual history revealed menarche at age 16 with regular cyclic periods until menopause at age 46 followed by no further bleeding.

The physical examination revealed a well developed but emaciated white female. Body weight was 84 pounds with height of 60½ inches. Blood pressure was 115/70, pulse 108, temperature 98.6°F. Positive findings were a dark beefy tongue and a small, oval, hard, non-tender nodule on the right side of the neck. Chest examination revealed the right diaphragm to be fixed. Moist, harsh rales were heard in both bases posteriorly. There was dullness to percussion and reduced breath sounds in both bases. Abdominal examination revealed distention. A fluid wave was elicited. Venous congestion was present on the abdominal wall and lower thorax. The abdomen was tympanitic anteriorly but dull in the lateral areas. There was extreme tenderness in the right upper and left lower quadrants with a hard, irregular, fixed tender mass in the left lower quadrant. There was also some tenderness in the left costovertebral angle. The skin was dry, and there was a two-plus pitting edema of the left ankle and pretibial area. Pelvic examination revealed a frozen pelvis.

Laboratory examination showed the hemoglobin to be 11.6 grams; white blood count was 13,000 with a normal differential; VDRL positive twice with 32 Kahn and 32 Kolmer units; NPN 39 mgm. per 100 ml.; creatinine 1.5; sugar 117; sodium 133 mEq./liter; potassium 5.5 mEq./L.; CO_2 22.7 mEq./L.; chlorides 99 mEq./L.; total protein 5.9 Gm./100 ml. with 1.78 Gm. globulin and 4.12 Gm. albumin; sedimentation rate 23 millimeters per hour. EKG and x-rays will be shown.

Hospital course: The patient was admitted to the hospital on February 1, 1954. Paracentesis with removal of 2000 cc. was done on the following day. Pelvic examination after paracentesis revealed the vagina and cervix negative aside from a slight bloody discharge from the cervix. The uterus was not definitely outlined but seemed fixed by a hard nodular tumor mass filling the pelvis.

The patient rapidly became worse and expired at 6:05 a. m. on February 4, 1954, the third hospital day.

Dr. Delp: Any questions of Dr. Harvey?

Q: Were any other blood pressure readings recorded?

Dr. Harvey: The blood pressure reported here is 115/70. That was the admission blood pressure. No other blood pressures were noted in the chart. A paracentesis was done on February 2. The pulse rate was then 140. The tachycardia continued on the 3rd and 4th.

Q: What did the paracentesis fluid look like?

Dr. Harvey: The fluid had a cloudy yellowish appearance.

Q: Was the fluid examined for cells?

Dr. Harvey: It was sent to the pathology laboratory.

Dr. Delp: The report read, "A large number of type 5 cells." Any other questions?

Q: Was there any tenderness in the left leg?

Dr. Harvey: No tenderness in the leg was mentioned.

Q: How long had the patient had the chronic cough and sputum?

Dr. Harvey: Her main symptoms had started two months before and fairly rapidly became worse. However, she had worked until December.

Dr. Delp: There is no specific reference either in the history taken in the clinic or on three histories in the hospital as to how long the patient had had this cough. I gathered from other notes that she had had the cough about two months.

Q: What was her actual red blood count?

Dr. Delp: 4,380,000.

Q: Can you elaborate on the abdominal symptoms?

Dr. Delp: After eating a very small amount, the patient felt diffuse abdominal discomfort. She couldn't exactly localize it. She had the discomfort even with taking water.

From the University of Kansas Medical Center. Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, obstetrics, radiology, pathology, and the third and fourth year classes of medical students.

Q: What were her bowel habits?

Dr. Harvey: No change. She had always been somewhat constipated. However, she did state that in recent weeks it felt as though she was having difficulty with her bowel movements.

Dr. Max Allen, associate professor of medicine: Can we assume that the parts of the physical examination not mentioned were negative?

Dr. Delp: Yes. This patient had a high degree of deafness, and it was rather difficult to communicate with her.

Q: What were the urinary findings?

Dr. Delp: Specific gravity 1.025, acid reaction with heavy trace of albumin, negative sugar, 10 to 20 pus cells per high power field. After paracentesis, a note on the chart states that the abdomen was somewhat softer and that the liver, spleen, and kidneys were not palpable.

Q: Could you elaborate on the patient after she became worse in the hospital?

Dr. Harvey: The pulse rate, as I mentioned, went up to rapid levels, the respirations increased, and the temperature gradually went down to subnormal levels. Dr. Hunter mentioned that he felt she was semicomatose.

Q: Was her blood pressure taken when she had a pulse of 140?

Dr. Harvey: No.

Q: Was she jaundiced?

Dr. Delp: No, nor had she been jaundiced at any time in her past.

Q: Was there any hemoptysis during her hospital course?

Dr. Harvey: No.

Q: Was there any chest pain?

Dr. Delp: No chest pain. She was quite dyspneic before death but had no chest pain. She was specifically questioned about it and denied any chest pain. This patient was complaining only of gastrointestinal symptoms. Any other questions? We have an EKG and x-ray films. Dr. Dimond, do you have any comments about the EKG?

Dr. E. Grey Dimond, professor of medicine: I think it is a normal EKG other than the sinus tachycardia. The Q waves are probably normal Q waves. The T wave is a bit broad. I read this as sinus tachycardia.

Dr. Delp: All right, thank you. Mr. Nystrom.

Mr. Curtis Nystrom, fourth year student:* We have a chest x-ray which was taken on the second day of hospitalization. The lungs are not congested in the apices. The angles are clear. There seems to be quite a bit of infiltration in both lower lobes with even more infiltration on the right side, which may be a little bit of bronchiectasis. The diaphragm is elevated, probably by fluid in the abdomen. The aorta is not widened. There does not seem to be any bone pathology. We don't see air under the diaphragm. The left lateral film doesn't add a whole lot. It does show some patchy infiltration in the lower part of the lung. The heart isn't enlarged.

Flat plate of the abdomen shows a haziness from the fluid. The bowel loops seem higher than usual as if they were displaced from the pelvis. I wouldn't definitely say what was holding the bowel out of the pelvis. It could be either some mass in the colon or a uterine or even an ovarian mass.

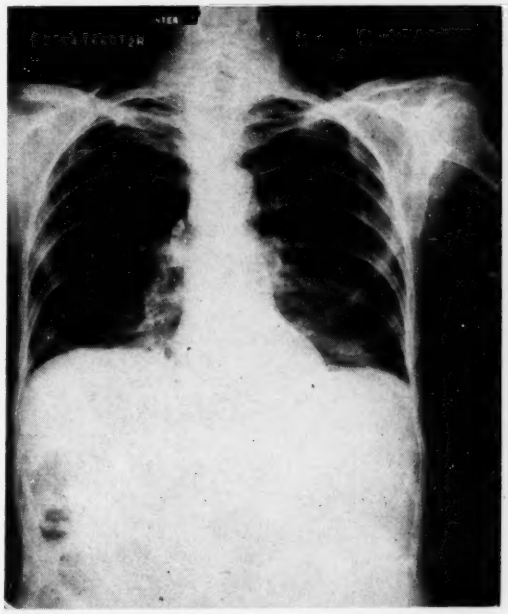


Figure 1. Chest x-ray showing elevated diaphragm and patchy atelectasis of lower lobes.

Dr. Delp: Dr. Tice, do you have any comments concerning this interpretation?

Dr. Galen Tice, professor of radiology: Dr. Litton's impression was that the changes in the chest were likely due to the high diaphragm with segmental or patchy atelectasis. I think the interpretation of the the abdomen is entirely correct.

Dr. Delp: We have a 59-year-old patient who presented herself to the outpatient department. We assume this patient walked in, gave her history, was examined, was referred to another clinic within the outpatient department. Then only a matter of a few days later she re-entered the hospital and within three days was dead.

The patient complained chiefly of gastrointestinal symptoms, discomfort, probably cramp-like pain, throughout the abdomen, precipitated by eating or drinking. She had some loss of appetite. She was able to eat only small amounts of food at a given

* Received M.D. degree, June 7, 1954.

time, because eating precipitated discomfort. A discomfort of distention in the rectum annoyed the patient considerably. She had a cough and had been bringing up a large amount of sputum—we're not certain for how long but at least for a couple of months. Her weight loss may be a little unreliable as far as the patient's recorded history is concerned.

At the time she entered the outpatient department her weight was recorded as 84 pounds. This patient had a lump in the neck removed in 1935. She said she now had a recurrence of a lump in her neck. Whether or not it was actually recurrent, I am not sure. It might be an entirely new lesion. After she entered the hospital, she had a paracentesis. So called type V cells were detected in this fluid. We need to explain what happened in such a relatively short period of time. Spearman, may we have your differential diagnosis?

Figure 2. Flat x-ray film of the abdomen showing haziness due to fluid and displacement of bowel loops from pelvis.

DIFFERENTIAL DIAGNOSIS

Mr. Jesse Spearman, fourth year student:* We have here today the case of a 59-year-old white female with the chief complaints of flatulence, eructation, and distention of the abdomen for two months duration and a weight loss for eight months. As Dr. Delp said, her weight at the time she came in was 84 pounds. That is pretty small. I could assume that she probably was small from the start of her symptoms. She also had a mass in the abdomen with unilateral edema of the leg. She was not complaining of pain. She had 32 Kolmer units and a positive VDRL. She had a distended abdomen, characteristic of ascites. I wish to base my differential diagnosis on the causes of ascites with a mass in the pelvis.

First, I'd like to consider the cardiac diseases. According to Cabot, 50 per cent of all ascites is caused by cardiac diseases. In this case we had no symptoms or physical findings that would lead me to believe that she had a cardiac lesion. I think on the basis of the history and physical findings, we can rule out cardiac disease as the cause of this patient's ascites.

The next I'd like to consider is glomerulonephritis. This is a common cause of ascites, but usually in glomerulonephritis individuals have presenting symptoms of edema elsewhere before complaining of ascites.

Liver disease is a common cause of ascites. You have to consider cirrhosis. This individual probably didn't eat very well. She could also have had a specific infection of the liver by syphilis.

Tuberculous peritonitis is another common cause of ascites. Tuberculous peritonitis in children is the commonest cause of ascites, but here x-ray examination was negative for pulmonary tuberculosis. In order to diagnose tuberculous peritonitis, I'd expect to have findings in the x-rays positive for tuberculosis.

Another consideration is neoplastic peritonitis. In patients of middle life or past, metastasis to the peritoneum is a common cause of peritonitis.

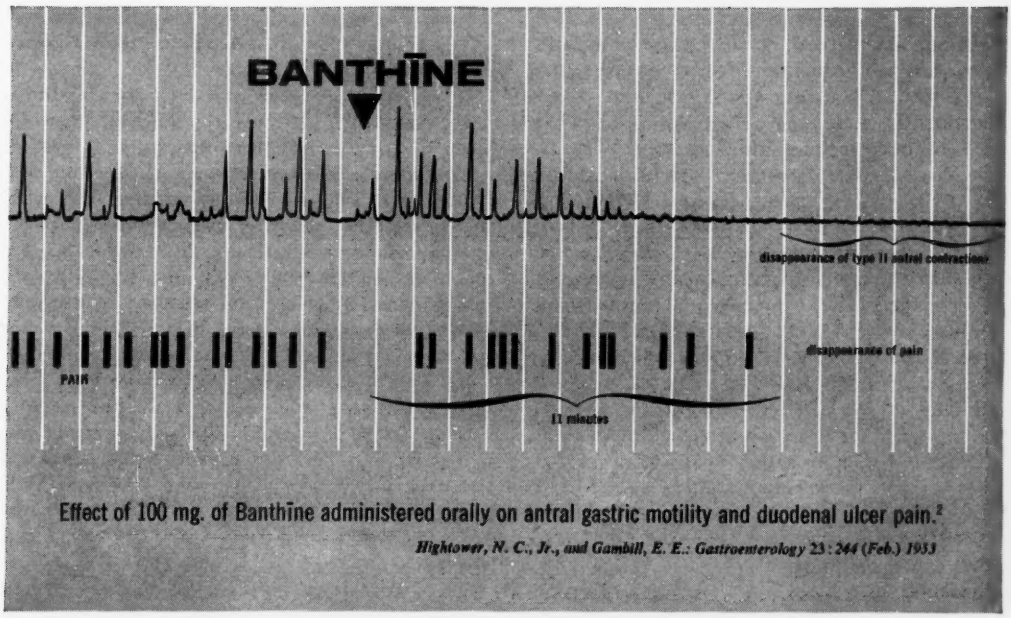
Nutritional edema could cause ascites. I think, if this were a nutritional edema, the serum protein reports in this case would be different. The total protein was 5.9 with a good albumin-globulin ratio.

Hemoperitoneum might be considered. One of the most frequent causes of hemoperitoneum is a rupture of ectopic pregnancy. This patient was 59-years-old and past the menopause, so I don't think she could have an ectopic pregnancy.

Other considerations are severe anemia, pernicious anemia, aplastic anemia, leukemia, or Hodgkin's disease. The laboratory and physical findings in this patient do not suggest these diseases.

Last I'd like to consider ovarian neoplasm. Ovarian cysts will give dullness in the midabdomen. Here tympany of the midabdomen was found. Ovarian fibroma is one of the rarities, but if you never think of it you'll never make the diagnosis. Very frequently ascites occurs and even hydrothorax. Another ovarian neoplasm I'd like to consider is a metastatic neoplasm referred to as Krukenberg's tumor. Krukenberg's tumor can have an origin in any place in the gastrointestinal tract: in the stomach, large intestine, small intestine, or even the gallbladder. If I were to consider Krukenberg's tumor seriously, I'd think mostly of a metastasis from the gallbladder, but in this patient there was no history of any gallbladder disease.

* Received M.D. degree, June 7, 1954.



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Hightower, N. C., Jr., and Gambill, E. E.: Gastroenterology 23:244 (Feb.) 1953

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Ruffin, J. M.; Texter, E. C., Jr.; Carter, D. D., and Baylin, G. J.: J.A.M.A. 153:1159 (Nov. 28) 1953.

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Carcinoma of the ovary includes the functional neoplasms. Ten per cent of all the solid neoplasms of the ovary are granulosa cell or theca cell tumors or luteomas. These particular neoplasms appear earlier in life, usually before the menopause. There have been cases reported after the menopause, but they are rare. There are other neoplasms such as pseudomucin or serous cyst adenoma. I don't think the physical findings of a hard nodular mass fit these.

In conclusion, I think that the primary diagnosis is secondary ovarian neoplasm from some place in the gastrointestinal tract, probably the sigmoid colon. Neoplasm of the sigmoid colon will give unilateral edema of the legs. There are many other questions that probably need answering. The stasis or the immobility of the diaphragm on the right side is one. Regarding the distention of the abdominal veins, I think this particular patient could very well have had more than one disease. With carcinoma of the sigmoid there could have been thrombosis of the portal system which would produce back pressure and the distention of the abdominal veins. Likewise, the hemorrhoids that have been reported could occur.

Dr. Delp: Thank you, Spearman.

Dr. Hunter, associate in gynecology and obstetrics: A fair amount of the patient's light weight was made up of fluid in the abdomen. Anyone who has a mass in the pelvis with ascites and a history like this patient gives probably has a pelvic malignancy. By that I mean ovarian malignancy. It might be noted that the main part of the nodular mass in the pelvis was on the left side. We interpreted that to be massive enough to impair circulation returning from the left leg, and we accounted for edema on that basis. Regarding the possibility of Krukenberg's tumor that has been mentioned, it's stated that Krukenberg's tumors are usually bilateral. They tend to retain the shape of the ovary, and that was not present in this case. No definite ovarian mass as such could be felt in this patient, even after the paracentesis, so I think that that would be against Krukenberg tumor. I think that the gastrointestinal complaints she had probably were secondary to ascites and were not primary. As for the beefy red tongue, she gave a poor dietary story, I think. It was difficult to talk to this patient because she was hard of hearing and very sick.

Our diagnosis at the time was ovarian malignancy with carcinomatosis, ascites, and probable pleural implant or pulmonary metastasis in spite of the negative chest x-ray. I think the terminal event was bronchopneumonia. This patient's terminal course is not very well documented, as you readily see. From what we know about it, the patient had physical signs of some pulmonary pathology on admission, and her course in the hospital was a steadily worsening one. She did tend to vomit near the end. I think that probably aggravated it, and I would be much in favor of bronchopneumonia as the terminal event causing her death.

Dr. Delp: Dr. Wheeler, you did make a diagnosis in the outpatient department of left ovarian malignancy. Would you document that for us?

Dr. John H. Wheeler, assistant clinical professor of medicine: The diagnosis, of course, was made on the fact that she was obviously chronically ill and she had an easily palpable mass in the abdomen and ascites. Pathologically, we could assume that she had peritoneal implants, and we came to that conclusion because of the absence of other findings. She was sent to gynecology clinic that same day.

Dr. Delp: I'm just never satisfied with this diagnosis of malignancy as being the cause of death. I know I belabor this until it must annoy some of you, but there must be another explanation as to why this patient died. I've seen patients live with intra-abdominal malignancies for two and three years. There must be an explanation for this patient's sudden death. Dr. Allen, do you have any ideas?

Dr. Max Allen, associate professor of medicine: Well, I think I'll go along with the student. I think unilateral edema of the leg with no known explanation for obstructive venous insufficiency of that leg would be a good reason to presume venous thrombosis and subsequent pulmonary embolus. I would like also to comment that the cough and excessive sputum as here described, even with negative chest films, can occur with diffuse lymphogenous metastasis to the lung, with carcinoma of the stomach being perhaps the most common primary site. I think the terminal event must have been a pulmonary embolus. I realize that withdrawal of ascitic fluid with depletion of body fluid and electrolytes can be the reason for a low sodium syndrome developing, producing death by the depletion of circulating blood volume. I would think there would be some other indication of that before the sudden death of the patient.

PATHOLOGY REPORT

Dr. Hans Peters, resident in pathology: The patient appeared remarkably older than her given age. Though we saw the little nodule on the neck, it was not our impression that it was anywhere near the phrenic nerve. It was much higher up, and unfortunately we were not able to take a section of it. We had the impression it was a fibroma.

The abdomen of this patient was markedly distended, and when it was opened we were able to obtain 3500 cc. of serosanguineous fluid. Some of the loops of the bowel were discolored, black bluish red, and there was fibrinous exudate over the entire mesentery and peritoneum.

First of all, I'd comment on the mass. This was found in the left lower quadrant and in the left

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pelvis. It was about 13.5 cm. by 12 cm. by 6.5 cm. It could very well have compressed the left iliac vein. It was connected to the uterus by direct extension by the tube, which split the tumor into two halves. The lower part was placed in the pelvis, and

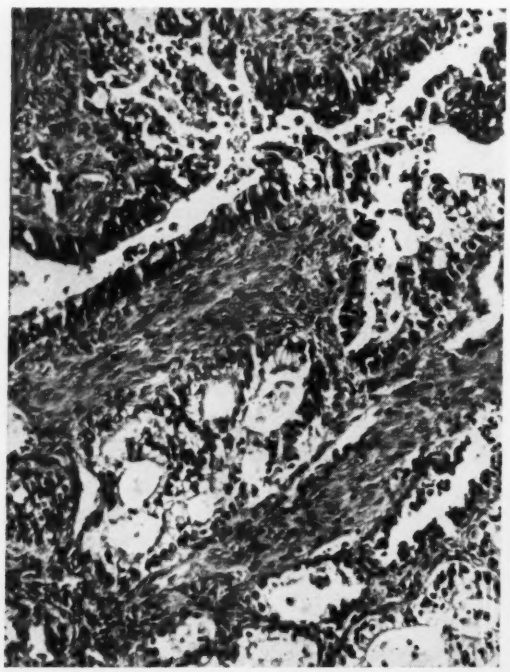


Figure 3. Hematoxylin and eosin stained section of adenocarcinoma of the left ovary showing mucus-producing glands, particularly in the lower portion.

the upper part was above the tube and more in the lower quadrant. The uterus itself was not markedly enlarged. There appeared to be some necrosis of the endometrium of the right tube and of the right ovary.

To look for a cause of the discoloration of the bowel, careful dissection of the superior mesenteric artery was done. A thrombus formation was found in branches of the superior mesenteric artery with infarction of two-thirds of the bowel, as far as to the ileum. The aorta showed marked arteriosclerotic changes with a few ulcerations. On one of the ulcerations, which was just about the iliac axis, thrombus formation was noted. This might very well have been the cause for thrombosis of the superior mesenteric artery. There was infarction of the left kidney and infarction of the spleen. Both of them were small.

The findings in the lungs were restricted to the slides, probably a pneumonic process.

Dr. Harlan I. Firminger, associate professor of pathology and oncology: I have only a little to add from the histological examination to what Dr. Peters has already told you. Sections of the ovarian tumor all showed a glandular type of carcinoma, with tall columnar cells and some pseudostratification, and in some areas there were actually papillary-like projections.

In other areas the adenocarcinoma cells had clear cytoplasm with basally located deeply staining nuclei, and the cytoplasm appeared to be filled with a mucinous-like material. Typical signet cells were evident in many areas.

A PAS stain showed the mucin content of these cells in the tumor. Actually, mucinous material was also evident grossly.

Section of the myometrium revealed small glandular structures infiltrating the myometrium throughout its entire depth, including invasion of venules. There were no solid masses of tumor anywhere in the uterus. The endometrium was thoroughly necrotic over a wide area for some peculiar reason, and there was apparent thrombosis of vessels. This appeared to be infarction of the endometrium of a rather diffuse nature.

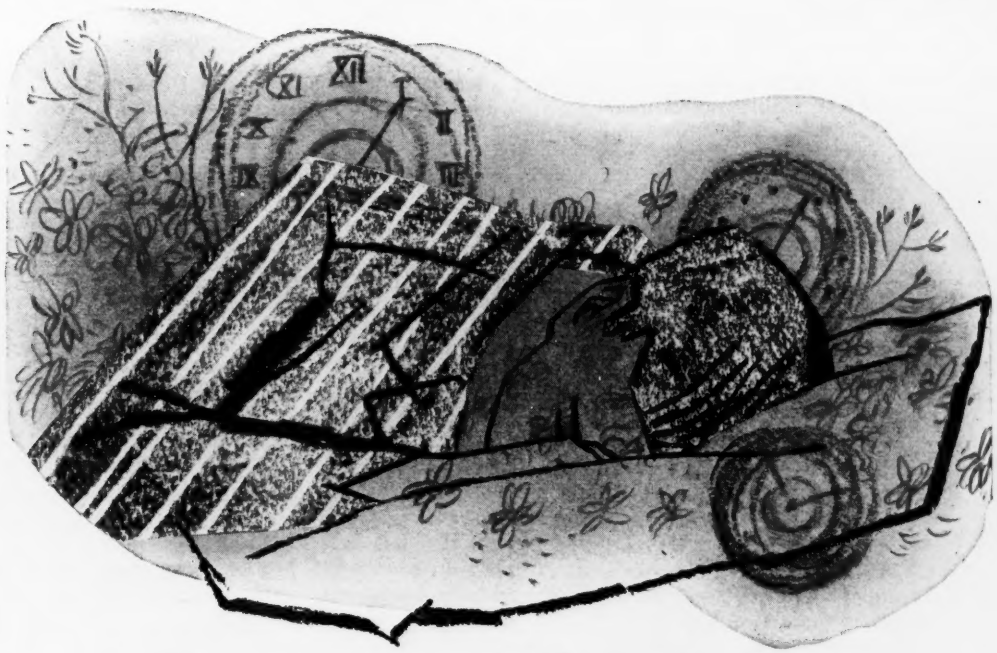
The liver showed some periportal fatty change, pretty much restricted to the portal areas.

Figure 4. Hematoxylin and eosin stained section of mesenteric artery containing recent embolus showing beginning polymorphonuclear infiltration in the muscular coat of the artery.

Section of the aorta in the area of ulceration above the origin of the celiac axis revealed ulceration of a tremendous atherosclerotic plaque.

The kidneys showed recent infarcts.

The superior mesenteric artery contained recent



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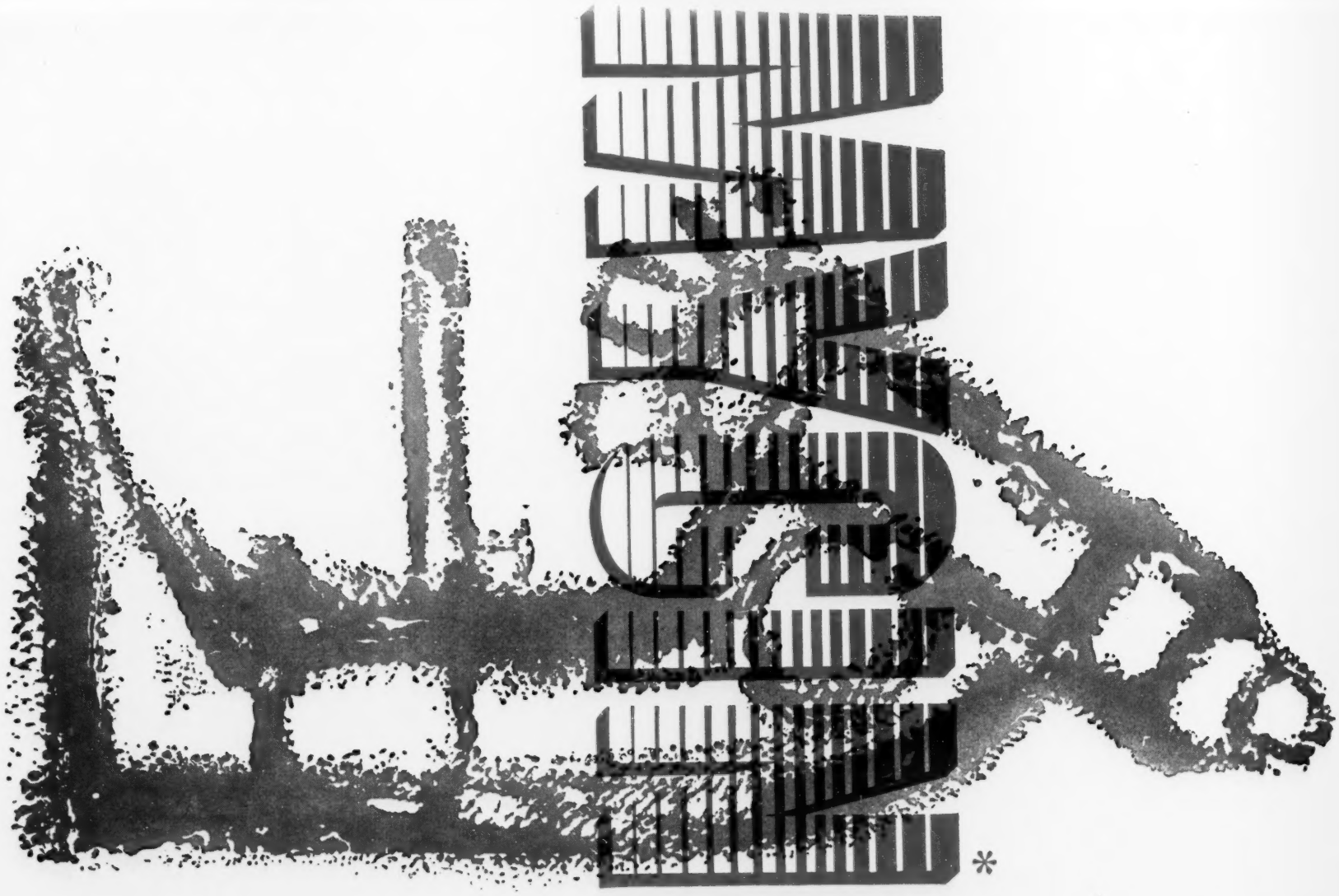

thrombotic material. Multiple sections revealed what looked like a little fibroblastic content in some areas. A few fibroblasts were suggested, but they were questionable microscopically. That the thrombosis was not post mortem was quite evident because of the polymorphonuclear infiltration in the media of the vessel.

In summary, this is a patient who presented herself with a primary ovarian carcinoma of the left ovary which had not metastasized widely. We found no evidence of metastases with the exception of the extension in lymphatics and interstitially in the uterus immediately adjacent to the tumor. Actually one would consider in the differential diagnosis the possibility that this arose in the uterus. However, its pattern in the uterus was very much against this, being just individual little glands here and there and in vascular structures. There was no isolated mass in the endometrium. Actually, if some one had told me this endometrium had been irradiated I would have believed it because it appeared like the necrotic picture of irradiated endometrium and thrombosis of vessels.

But knowing this was not done, it seemed that that must have been due to vascular obstruction and an unusual infarction of the endometrium, at least in some areas. Then, as Dr. Delp tried his best to point out, something happened to this patient in addition to malignancy, something entirely unrelated to the presence of malignancy unless emaciation and debility are important.

What role paracentesis and examinations may have had, I can't say. She did have arteriosclerosis of the aorta with ulceration and plaque formation. Within a few days, I would suggest four to seven days, before her death, I presume that a portion of the mural thrombus in the aorta broke loose and into several pieces. This may have occurred more than once because the lesions in the spleen and kidneys appeared slightly older. The lesion in the bowel, I would guess, happened two or three days ante mortem. These bits of mural thrombus broke loose and lodged in the arteries with orifices just below this area. This produced infarction. Subsequent symptoms were primarily due to the lesion in her bowel from acute infarction of the bowel. Shock developed. I'm sorry blood pressures weren't taken, but I'm sure she must have had shock level blood pressures. The pulse rates would bear that out. In addition, a certain amount of peritonitis occurred. How much it was related to paracentesis and how much was secondary to leakage through this degenerating bowel, I cannot say. I think it could be secondary to the infarction.

Dr. Delp: I'm completely unabashed in continuously championing the idea of studying all patients really carefully, sincerely, and conscientiously from a clinical standpoint. I don't mean that you have to subject such patients as this to all sorts of gadgeteering to examine them, but I think you should do a meticulous physical examination. I think you should be thoughtful about the findings, and I'm particularly talking about patients with malignant disease. It is hoped that some time in the future we will be handed some sort of substance or some sort of method whereby we can cure malignant disease. I think a good many clinicians are really going to be in a bad way with the fatalistic attitude. The presence of neoplastic disease is not a satisfactory nor complete explanation for death in many cases—herein is such a case.

ANATOMICAL DIAGNOSIS

Primary

Arteriosclerosis of the cerebral and coronary arteries, slight, with one area of almost complete occlusion in the anterior descending branch of the left coronary artery; arteriosclerosis of the aorta, advanced, with ulceration of the abdominal aorta 2 cm. superior to the origin of the celiac axis.

Recent embolic occlusions of the superior mesenteric artery and a branch of the splenic artery.

Recent hemorrhagic infarction of the jejunum and ileum and small hemorrhagic infarcts in the left kidney and spleen.

Acute dilatation of the stomach, duodenum, and first part of the jejunum with partially digested food and brownish-green material, estimated 2000 ml. volume.

Multiple acute erosions of the mucosa of the stomach.

Acute fibrinous peritonitis with localized abscess formation adjacent to the descending colon 10 cm. below the splenic flexure.

Serosanguineous fluid in the peritoneal cavity, 3,500 ml.

Bronchopneumonia of the lower lobes of both lungs, moderate.

Gastric contents and mucus in the treachea and in both main bronchi.

Recent unhealed puncture wound in the lower anterior abdominal wall (history of paracentesis two days before death).

Recent unhealed puncture wounds in the antecubital fossae (history of venipuncture three days before death).

Adenocarcinoma of the left ovary with fibrous adhesions to the left tube, pelvic wall, and to part of the ileum, and extension of tumor into the uterus and invading small vessels in the myometrium.

Thrombosis of veins in the myometrium with necrosis of endometrium.

Submucous leiomyomas in the wall of the vagina and in the uterus.

Hypertrophy and dilatation of the urinary bladder

who have seborrheic dermatitis of the scalp

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Published reports on more than 400 cases[1-8] show that SELSUN completely controls seborrheic dermatitis in 81 to 87 percent of all cases, and in 92 to 95 percent of common dandruff cases. It keeps the scalp free of scales for *one to four weeks*—relieves itching and burning after only two or three applications.

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1. Slepyan, A. H. (1952), Arch. Dermat. & Syph., 65:228, February. 2. Slinger, W. N. and Hubbard, D. M. (1951), *ibid.*, 64:41, July. 3. Sauer, G. C. (1952), J. Missouri M. A., 49:911, November.

1-348-54

with trabeculation, slight (history of nocturia for unspecified period).

Emaciation (history of 15 pounds weight loss over a period of eight months).

Brown atrophy of the heart, moderate, weight 230 gm.

Accessory

Foci of fibrosis in the myocardium.

Bullous emphysema of the middle and upper lobe of the right lung and the upper lobe of the left lung, moderate.

Fibrous pleural adhesions at the apex of both lungs and in the posterior region of the lower lobe of the right lung.

Calcified lymph nodes at the hilus of the right lung, substernally, near the right first interspace, and beside the aorta near the bifurcation of the trachea.

Cortical adenoma of right adrenal, small.

Retention cyst of the lower pole of the left kidney.

Focal area of fibrosis in the petrous portion of the temporal bone (history of measles followed by deafness 56 years before death).

Subcutaneous nodule in the right lateral region of the neck.

Atrophy of both breasts, moderate.

Hemorrhagic areas over both patellae.

Edentia.

COUNTY SOCIETIES

At a meeting of the Sedgwick County Society held on May 11 the following officers for 1955 were elected: president, Dr. George E. Cowles; vice-president, Dr. Ernest W. Crow; secretary, Dr. Charles P. McCoy; treasurer, Dr. Don E. Miller; censor, Dr. Lennel I. Wright; three-year terms on the board of directors, Dr. Leo K. Crumpacker, Dr. Harold F. O'Donnell, and Dr. Charles L. Woodhouse.

A joint meeting of the Bourbon County Society and the Mercy Hospital staff of Fort Scott was held at Fort Scott in May. Dr. Rollin R. Nevitt reported on the annual meeting of The Kansas Medical Society.

Dr. Charles Hunter, associate in obstetrics and gynecology at the University of Kansas Medical Center, was guest speaker at a meeting of the Labette County Society held at Parsons in May. He discussed new mechanical devices used in his specialty.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

THE A.M.A.

Some people seem to have the idea that the American Medical Association, as the representative of the medical fraternity in this country, does little or nothing except fight socialized medicine—that, in short, it takes a negative, not a positive, attitude towards medical problems.

The Louisville, Ky., *Courier-Journal* decided to find out just what does go on in A.M.A. headquarters. It sent a staff writer, Robert Clark, to investigate. And it featured a long article by him which contains facts of interest to us all.

Some 60 per cent of all the association's revenues are spent for scientific work. It prints nine medical journals, scores of books, and thousands of pamphlets, all devoted to the war against disease and related matters. It conducts extensive laboratory tests in the fields of chemistry, microbiology and physical medicine. It analyzes widely used products, ranging from foods to cosmetics. It rates hearing aids and other devices for acceptability. It maintains files on illegal practitioners, quacks, and fake and dangerous medicines. One of its newest activities, begun in 1952, has to do with the ever-increasing problem of mental health. A program of study in nine mental health fields is going on. It sponsors a foundation which has given close to $5,000,000 to the nation's medical schools since 1951. It maintains medicine's largest reference library.

So it goes, in Clark's vivid and factual article. And at the end he quotes this significant paragraph from a statement made by an A.M.A. official: "Our most urgent effort should now be directed to the solution of the problem of the medically indigent and the chronically ill. We believe that this objective can be reached without major change in our existing mechanism."—*Atchison Globe, May 27, 1954.*

FAMILY DOCTOR STILL ON THE JOB

The claim has often been made that the practice of medicine has developed into specialization. That

for greater safety in streptomycin therapy...

DISTRYCIN

Squibb Streptoduocin
Streptomycin and dihydrostreptomycin in equal parts

Distrycin has an important advantage over streptomycin. It has the same therapeutic effect but ototoxicity is greatly delayed. Since the patient is given only half as much of each form of streptomycin as he would have on a comparable regimen of either one prescribed separately, the danger of vestibular damage (from streptomycin) or cochlear damage (from dihydrostreptomycin) is significantly lessened.

Signs of vestibular damage appear in cats treated with Distrycin as much as 100 per cent later than in animals given the same amount of streptomycin.

Cat treated with streptomycin shows no nystagmus after whirling.

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On dosage of 1 Gm. per day for 120 days, ototoxicity was as follows*:

	Vestibular damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	12	6	18
Dihydrostreptomycin	6	0	6
Distrycin	0	0	0

	Cochlear damage % of patients		
	Mild	*Moderate*	*Total*
Streptomycin	0	0	0
Dihydrostreptomycin	12	3	15
Distrycin	0	0	0

Heck, W.E.; Lynch, W.J., and Graves, H.L.: Acta oto-laryng. 43:416, 1953.

Distrycin dosage is the same as for streptomycin. In tuberculosis the routine dose is 1 Gm. twice weekly, in conjunction with daily para-aminosalicylic acid or Nydrazid (isoniazid). In the more serious forms of tuberculosis, Distrycin may be given daily, at least until the infection has been brought under control.

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the family doctor had disappeared, much to the detriment and discomfort of the average family. The family doctor has always been in the front line of defense against illness, and has won for himself a secure place in American hearts. Always ready and willing to answer calls day or night, he was more concerned with doing good than in making money.

In addition to having a broad knowledge of all of the physical aspects of disease the family doctor was usually a counselor, confidant and friend. His services went far beyond the practice of his profession, and he became a ready help in time of trouble, sickness and otherwise.

Now it seems the family doctor is still on the job, despite these rumors. Some 4,000 of them, gathering from all parts of the nation, met in Cleveland recently under the auspices of the American Academy of General Practice. For three days those in attendance discussed every phase of a general practice necessary to meet the medical, emotional and spiritual requirements of his patients. He must keep abreast of new discoveries in the field of pathology, that those in need of special treatment can be advised in time.

It was brought out at the meeting that the physical health of the American people is better than their emotional health, as indicated by the fact that 18 tons of aspirin are consumed every day in the United States. The family doctor is still on the job, and the nation will be all the better for it.—*Wichita Eagle, April 3, 1954.*

BOOK REVIEWS

Mayo Clinic Diet Manual, Second Edition. By Committee on Dietetics, Mayo Clinic. Published by W. B. Saunders Company. 247 pages. $5.50.

In reviewing this manual we have found it to be somewhat disappointing. The material is presented in what is generally considered to be an outmoded method. That is, the diets are classified according to the specific condition for which they are to be used. This seems to imply that for each condition discussed these are the only diets which can be used and that these diets cannot be used in other conditions unless repeated in those sections. This type of presentation is misleading to those who use the manual as reference in that not all the diets which may be used can be found in the proper section (e.g., high or low protein levels are found only in the section on children's diets). Also, not all conditions for which dietary treatment may be necessary can possibly be listed.

This manual includes some materials which we would consider to be superfluous, such as the use of a table of caloric values.

It is also a little surprising to find that the dietary regime for diabetic patients advocated by the American Diabetes Association and the American Dietetic Association has not been adopted by the Mayo Clinic, though this is not of necessity the fault of the authors of this manual.

The information given on each specific diet is complete, with the approximate composition of each diet given along with a discussion of the adequacy of the diet. The types of foods to be included and excluded are given, as well as the dietary pattern and sample menu. We do feel, however, that there would be a distinct advantage in having the dietary pattern given first, followed by the foods to be included and excluded. Otherwise, the outlines for each diet appear adequate and clearly outlined.—*E.M. and A.P.*

A Doctor Talks to Women. By Samuel Raynor Meaker. Published by Simon and Schuster, 630 Fifth Avenue, New York. 231 pages. Price $3.95.

Dr. Meaker has written a comprehensive, readable book on the functioning of the female body. This book could be a useful reference in college-level classes in hygiene, marriage-counseling, etc., and should be readily understood by the average intelligent adult woman. It includes definitions of medical terms.

Chapters 9, 10, and 11, titled respectively, "Conception and The Beginning of Pregnancy," "Fertility and Sterility," and "Help for Childless Couples" are especially rewarding and should lead sterile couples to a better understanding of their plight and what may be done to remedy it. The chapters on cancer, tumors and operations will also have a reassuring effect upon the reader.—*C.B.*

Hospital Service in U. S.

Hospitals set a new record during 1953 with 19,869,061 patient admissions as compared with 18,914,847 in 1952, according to the 33rd annual report on hospital services prepared by the A.M.A.'s Council on Medical Education and Hospitals.

For the second time in history, the report states, the number of hospital births exceeded the three million mark, 3,307,182, representing approximately 84 per cent of the estimated 3,910,000 annual births in the United States.

The A.M.A. registered 6,840 hospitals, 4,704 as non-governmental. Gains in patient admissions were noted in both groups but were more pronounced in the non-governmental division credited with 74 per cent of all admissions.

Bed capacity in registered hospitals increased by

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31,399 over the previous year—1,113,004 in governmental and 460,010 in non-governmental hospitals. Average length of stay per patient was reduced in general hospitals from 9.8 to 9.3 days. Federal general hospitals also showed a reduction—from 26.2 to 23.6 days.

Eight hundred fifty hospitals are approved for internships, 1,146 for residencies, and 1,071 for professional nursing education.

ANNOUNCEMENTS

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for 1954-1955. An award of $100 will be given the author of the best unpublished contribution on proctology or allied subjects. Competition is open to all physicians. Information may be secured from the Academy, 43-55 Kissena Boulevard, Flushing, New York.

The annual convention of the National Society for Crippled Children and Adults will be held at the Hotel Statler, Boston, November 3-5.

The 19th annual congress of the United States and Canadian Sections of the International College of Surgeons will be held in Chicago, with headquarters at the Palmer House, September 7-10. Information may be obtained from the College, 1516 Lake Shore Drive, Chicago 10, Illinois.

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in cooperation with the respective state chapters of the college as well as the staffs and faculties of the local hospitals and medical schools, will sponsor the 9th Annual Postgraduate Course in Diseases of the Chest at the Hotel Knickerbocker, Chicago, October 18-22. A similar course at the Hotel New Yorker, New York City, will be November 8-12. Tuition for each course is $75.

Details may be obtained from the College, 112 East Chestnut Street, Chicago 11, Illinois.

Schools of professional nursing in the United States admitted 43,327 new students in 1953, an increase of 1.8 per cent over 1952, it was announced recently by the Committee on Careers, National League for Nursing. Included in the group are students from three-year diploma programs and the four- and five-year collegiate programs.

In Kansas, where there are 26 schools of nursing, 641 students were admitted in 1952 and 604 in 1953.

All products advertised in the JOURNAL have been approved by the various councils of the American Medical Association.

In 1953, 21,400,000 Americans were injured in traffic accidents.

DEATH NOTICES

RICHARD G. MEISBURGER, M.D.

Dr. Richard G. Meisburger, 44, anesthesiologist at Bethany Hospital, Kansas City, died on May 26 after suffering a heart attack. A graduate of the University of Kansas School of Medicine in 1940, Dr. Meisburger interned at St. Luke's Hospital and was resident anesthesiologist at General Hospital, Kansas City, Missouri, before he was called into service in the Navy during World War II. He was an active member of the Wyandotte County Medical Society and of the Kansas City Society of Anesthesiologists. He was also a lecturer at the Kansas City University School of Dentistry.

CLARENCE E. BOUDREAU, M.D.

Dr. C. E. Boudreau, 76, El Dorado, died on June 14 as the result of injuries suffered in an automobile accident the day before. His first medical work was done at the Fort Worth School of Medicine, and he later studied at Columbia University College of Physicians and Surgeons, New York, from which he was graduated in 1913. He received his Kansas license the same year, and during his years of practice he specialized in surgery. He was a member of the Butler County Medical Society and a fellow of the American College of Surgeons.

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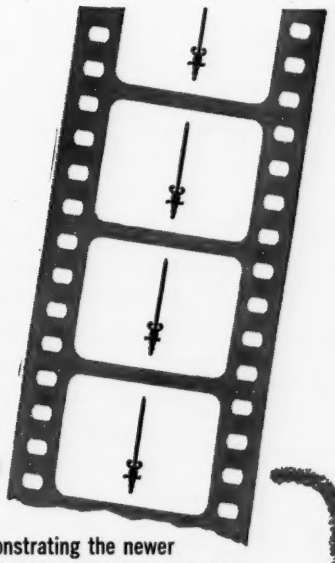

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Of the amount distributed so far, 2,930,818 cubic centimeters went to state health officials for use during the polio season. The balance, added to that being produced now, will provide a substantial supply for use in 1954.

A survey made recently for the *Journal of the American Dietetic Association* reports that Kansas physicians are making good use of material supplied by that group for simplifying and standardizing the diet of diabetic persons. Kansas was among the 10 states ranking highest in the list of those using the material.

The diet information was compiled from studies made jointly by the American Diabetes Association, the American Dietetic Association, and the Diabetes Branch of the U. S. Public Health Service.

In 1953, 38,500 Americans were killed in traffic accidents.

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If the patient complaining of aching joints is a woman between 37 and 54 years of age, it is highly possible that she is suffering from arthralgia rather than arthritis.[1] It has been estimated that arthralgia occurs in about 40 per cent of women with estrogen deficiency, and is exceeded in frequency only by symptoms of emotional or vasomotor origin.[2] In fact, arthralgia may be as indicative of declining ovarian function as the classic menopausal hot flushes.

Arthralgia, however, is just one of a vast number of distressing but ill-defined symptoms that may be precipitated by the loss of estrogen as a "metabolic regulator." Other good examples are insomnia, headache, easy fatigability, and tachypnea.

Because these symptoms sometimes occur years before or even long after cessation of menstruation, they are not always readily associated with estrogen deficiency, and the tendency may be to treat them with medications other than estrogen. Obviously, sedatives and other palliatives cannot be expected to produce a satisfactory response if an estrogen deficiency exists. Only estrogen replacement therapy will correct the basic cause of the disorder.

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1. Greenblatt, R. B., and Kupperman, H. S.: M. Clin. North America *30*:576 (May) 1946. 2. McGavack, T. H., in Goldzieher, M. A., and Goldzieher, J. W.: Endocrine Treatment in General Practice, New York, Springer Publishing Company, Inc., 1953, p. 225.

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It seems apparent that the general practitioner is going to be involved in the actual treatment of the tuberculosis patient to a much greater extent than he has in the recent past and fewer patients will be treated by specialists in chest diseases. Another factor acting in the same direction is the relatively shorter time that the average patient will spend in a hospital and the fact that such hospitalization is less likely to be in a remote institution. The family physician, therefore, will be less likely to lose supervision of his patient. This trend requires a change in the nature and emphasis of postgraduate medical conferences and courses of instruction, which must be directed more toward the general practitioner.—*James E. Perkins, M.D., NTA Bulletin, May, 1954.*

Among patients admitted to general hospitals, the prevalence of definite and suspected tuberculosis and of probably active tuberculosis is twice as high as among individuals x-rayed in mass surveys. Thus 14 (1.4 per cent) of every 1,000 patients show on admission x-ray the presence of definite or suspected tuberculosis as against 7 for every 1,000 in mass surveys. Two of every 1,000 x-rayed on admission to general hospitals show probably active tuberculosis as against one for mass surveys.—*1952 Annual Report, N.Y.S. Department of Health.*

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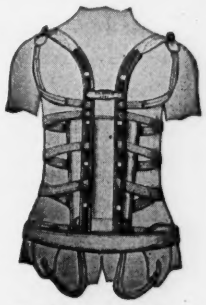

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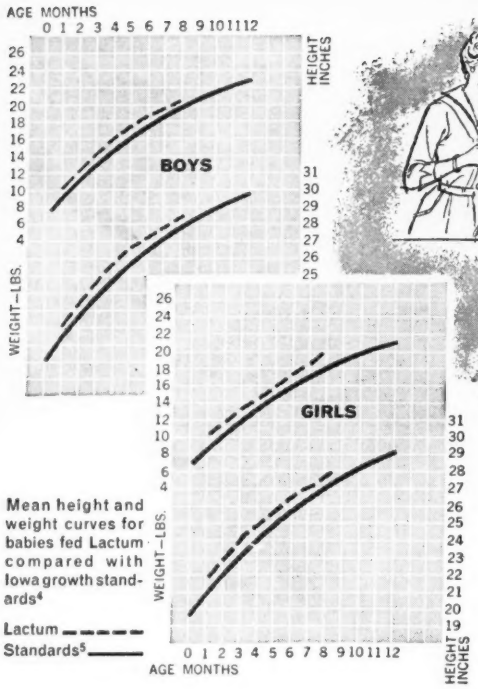
AGE MONTHS
0 1 2 3 4 5 6 7 8 9 10 11 12
BOYS
GIRLS
WEIGHT—LBS.
HEIGHT INCHES
Mean height and weight curves for babies fed Lactum compared with Iowa growth standards[4]
Lactum
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Lactum feedings are easy to prepare. One part of Liquid Lactum to 1 part of water, or 1 level measure of Powdered Lactum to 2 ounces of water, makes a formula supplying 20 calories per fluid ounce.

(1) Jeans, P. C.: In A.M.A. Handbook of Nutrition, Ed. 2, Philadelphia, Blakiston, 1951, p. 275. (2) Albanese, A. A.: Pediat. 8: 455, 1951. (3) Holt, L. E., Jr., and McIntosh, R.: In Holt Pediatrics, Ed. 12, New York, Appleton-Century-Crofts, Inc., 1953, pp. 175-178. (4) Frost, I. H., and Jackson, R. L.: J. Pediat. 39: 585, 1951. (5) Jackson, R. L., and Kelly, H. G.: J. Pediat. 27: 215, 1945.

Calculated on the basis of a daily allowance of 3.5 Gm. per Kg.

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